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THE PATHOGENESIS OF THE EDEMA OF CONGESTIVE HEART FAILURE*

E. H. BRESLER, M. D.

NEW ORLEANS

INTRODUCTION

In selecting the topic of this thesis my intention was to choose a commonly encountered clinical phenomenon, yet one concerning which there was some controversy in the literature. In so doing, I hoped to avoid a simple bibliographical survey, allowing myself the privilege of selecting only that portion of the literature which appeared to contribute toward a more rational viewpoint. As a result this thesis is admittedly biased, my only justification in this regard being a deep personal conviction concerning the essential validity of its contents.

The problem of fluid balance and renal function has long attracted my interest. The relation of this problem to that of congestive failure was made clear to me by an article on water and salt balance in congestive failure by Burch and Reaser.[1] My interest was further stimulated by an article on the fluid dynamics in congestive failure by Warren and Stead.[2] It then occurred to me that the problem of the pathogenesis of cardiac edema would be a desirable topic which would afford me the opportunity of familiarizing myself with one of the frontier fields of medicine about which our concepts are undergoing rapid change.

The foregoing thesis makes no pretense at an objective survey of the subject, but is written rather in the spirit of attempting to find an answer to one of the unsolved problems in medicine.

EDEMA

Edema is associated with a variety of pathologic conditions, one of the most commonly encountered of which is disease of the heart. It is the purpose of this paper to review the classical mechanisms relating failure of the myocardium with the dropsical state, and to discuss in some detail recent experimental evidence pointing toward a new and entirely different concept.

The term edema is most simply defined as an abnormal collection of fluid in the intercellular spaces. However, such a simple definition does not do justice to the variety of types encountered clinically. It is therefore important to differentiate between two major types of edema, generalized and localized. The distinction was made very clearly by Starling[3] in the eighth of a series of remarkable lectures published in 1912 under the title, "The Fluids of the Body", from which this excerpt is taken: "The term dropsy may be used to denote either an increased amount of fluid in the connective tissue spaces, especially of the skin, when it is sometimes designated as anasarca or oedema, or an accumulation of fluid in the serous spaces of the body, as in conditions of hydrothorax and ascites. In some cases the production of oedema may be brought about by local changes, and therefore in-

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volves merely a different distribution of the body fluid. Often however, the increase of fluid in the connective tissue spaces, including the serous spaces, is more or less general and may be associated with a simultaneous increase of the other chief fluid of the body, namely, the blood."

This distinction between the two types of edemas, while seemingly an obvious one, carries with it certain fundamental implications which are pertinent to the ensuing discussion. In considering localized edemas two common examples come immediately to mind, namely those following lymphatic obstruction and those following tissue injury, be it mechanical, thermal, or chemical. If one were to obstruct the lymphatic return in a limb or subject the limb to some type of trauma, edema will appear in this part even though no fluid was ingested by the organism throughout the experiment. Hence such edemas are not concerned with the balance between fluid intake and output. This is in sharp contradistinction to generalized edematous states wherein the total fluid content of the body is increased. Such a state must obviously be dependent upon a disproportion between the fluid intake and output of the organism.

The edema of congestive heart failure is characterized by an increase in the total fluid content of the body, sometimes attaining such proportions that the elastic limit of the confining skin membrane is exceeded and spontaneous rupture takes place. The apparent localization of this edema is an artefact produced by the action of the force of gravity, the greatest accumulations appearing in the most dependent regions of the body. Cardiac edema may be differentiated from the generalized edema of nephrosis and hypoproteinemia by virtue of the fact that together with the increase in interstitial fluid volume there is a simultaneous rise in blood volume as shown by Gibson and Evans,[4] Warren and Stead,[2] Seymour et al.,[5] whereas it has been pointed out by Warren, Merrill, and Stead[6] that blood volumes are essentially normal in the case of edemas associated with low serum proteins. Levine[7] used this feature to differentiate the edema of nephritis from that of heart failure in a patient. The blood volume in this patient was normal and subsequent studies on the dynamics of the circulation supported the fact that the patient did not have heart failure. Cardiac edema may be differentiated from the edema of acute nephritis due to the fact, as stated by Fishberg,[8] that the protein content of the edema fluid is lower in the former.

The prevailing concepts with regard to the mode of origin of edema, which have been in general acceptance for the past fifty years since Starling's original publication, have been based upon the factors affecting filtration and absorption of fluids at the capillary interface. Attention has been so strongly focused upon the microscopic capillary segment that the relations of fluid balance to the organism as a whole have been neglected. Reasoning from a theoretical consideration of the interplay of the various forces involved in the exchange of fluids across the capillary membrane, it is said that in heart failure, conditions arise which tend to increase the rate of filtration and decrease the rate of reabsorption. This in turn is then said to cause an efflux of fluid from the blood stream into the interstitial spaces resulting in edema. While it cannot be denied that such a shift of fluid might conceivably cause edema, one then wonders why the urinary output is diminished in patients with congestive failure. One might suggest that as a consequence of the fluid efflux the blood becomes concentrated, thus hindering urine formation. However, no such concentration of the blood has been demonstrated. In fact, Seymour et al.,[5] as well as others, have shown that in congestive failure there is a dilution of the blood as evidenced by low serum protein concentrations. This and many other observations cast considerable doubt upon the classical concepts of the mode of formation of generalized edemas, and suggest that a new approach to the problem be sought. However in view of the fact that Starling's concepts of edema formation in cardiac failure are, even now, so universally accepted,

a more critical examination of their validity is warranted.

According to the proponents of this theory, the edema of cardiac failure is due to one or a combination of three factors: (1) increased capillary permeability; (2) increased venous pressure; (3) hypoproteinemia. Each of these in turn will now be discussed in some detail.

INCREASED CAPILLARY PERMEABILITY

It has been suggessted, but as yet not proved (Landis[9]) that the prolonged slight anoxemia of cardiac decompensation may increase capillary permeability and be partially responsible for cardiac edema. That the capillaries would be so damaged in congestive failure is subject to grave doubt in view of the fact, as pointed out by Burch and Reaser,[1] that one sees no clinical evidence of edema in many severe anoxic states such as the tetralogy of Fallot or at high altitudes where the degree of anoxia is greater than that seen in congestive failure. Furthermore as pointed out by these authors, the protein content of the edema fluid in cardiac failure has been found to be in the neighborhood of 0.5 per cent, a figure too low to have any appreciable effect upon edema formation.

INCREASED VENOUS PRESSURE

The factor which is considered to be by far the most important in the pathogenesis of cardiac edema is the increased transudation of fluids taking place at the capillary interface as a consequence of elevated venous pressure. So much reliance has been placed upon this mechanism that if it can be definitely eliminated as even a contributing factor, the classical theories of cardiac edema formation must of necessity be abandoned.

A clear exposition of the purported role of elevated venous pressure in the genesis of cardiac edema has been advanced by Harrison[10] who states as follows: " As has been discussed, rightsided failure is practically always associated with a rise in the venous pressure. This results in a damming of blood in the capillaries and an eventual increase in the intracapillary pressure. Since there is no compensatory increase in the effective osmotic pressure of the blood, an increased filtration of water into the tissues will be the result. For a time this may be compensated for by a greater volume of lymph flow from the affected part.

However, as the venous pressure rises further, the faster rate of lymphatic drainage may not be sufficient to compensate for the increased rate of filtration and edema will result. Furthermore, if the venous pressure should rise still further, it will tend to interfere with the emptying of the thoracic duct." Harrison minimized the effects of increased capillary permeability.

This theory, because of its simplicity, is certainly tempting. However, it is based upon the assumption that failure of the right ventricle, in and of itself, will produce an elevated venous pressure. This assumption is not supported by direct experiment. In fact investigations designed to test the validity of this concept of "backward pressure" exerted by the failing myocardium have demonstrated clearly and irrefutably that the generalized increase of venous pressure is not directly caused by the failure of the heart as a pumping mechanism. Starr et al.[11] found that by severely damaging the right ventricle of the heart in dogs in both acute and chronic experiments, more than a minimal rise in venous pressure was never produced. In another series of experiments, Starr[12] found that after death the average pressure in the veins of cardiac patients who died with venous congestion, exceeded that of a series of controls who died without heart disease, by an amount approximately equal to the difference present during life. Since the elevation of venous pressure continues to be present even after the heart has ceased beating, it is quite evident that mechanical failure of the heart could have had no part in its production during life. The author ventures the opinion that the increase in venous pressure may be in large part accounted for by the enormous blood volumes which distended the vascular trees.

Further evidence disputing the role of increased venous pressure in edema forma-

tion was furnished by the experiments of Warren and Stead.[2] These investigators observed that in cardiac patients, in whom failure was precipitated by the administration of liberal salt diets, an accumulation of extracellular fluid, evidenced by an increase in weight, occurs before any increase in venous pressure is demonstrable. Associated with this gain in weight there is a proportionate increase in the blood volume, and only as a later manifestation does the venous pressure become elevated. Reasoning from the results of this and Starr's work, one is impelled to the conclusion that the elevation of venous pressure is determined by the overfilling and distention of the intravascular system with an excessively large blood volume.

There are many other facts which militate strongly against any possible role of venous hypertension in the causation of edema. Burch and Reaser[1] suggested the following objections: Ligation of a large vein, the inferior vena cava, just below the renal veins, will result in venous pressure values of six hundred or more millimeters of water pressure in the tributaries without the development of clinical edema in the areas drained. One might object that such a procedure will not affect renal function or impose an obstruction to the emptying of the large lymphatic trunks. These objections are overruled by the fact that in concretio cordis or mediastinal caval obstruction there is often marked venous hypertension for prolonged periods without clinical edema. Smirk[13] pointed out that the increased pressure produced by gravity far exceeds that seen in cardiac failure, so that in the upright position a short person with severe cardiac failure may have a lower venous pressure in the ankles than a tall person without cardiac failure.

These observations lead one to the conclusion that lymphatic reserve must be ample to suffice for even the largest transudations resulting from venous obstruction. There are however areas in the body where lymphatic drainage is not so efficient and where even its role in reabsorption is open to question (Peters[14]). These are the peritoneal and pleural cavities. Whenever excessive transudation as a result of venous hypertension takes place in these cavities, fluid readily accumulates. This fluid after a time becomes high in protein content assuming the characteristics of the edema of lymphatic obstruction. This would suggest that reabsorption is taking place through the blood capillaries rather than lymphatics. Therefore these transudates are to be considered as special types of localized accumulations of fluid incidental to the venous hypertension, which occurs only after a considerable degree of subcutaneous edema has already accumulated. The behavior of these isolated accumulations of fluid must then be interpreted separately from generalized edema.

HYPOPROTEINEMIA

It has been stated by Landis[9] that the general malnutrition due to the decreased appetite and impaired physiology consequent to congestion leads to a state of hypoproteinemia, thereby contributing to the formation of edema in the patient with chronic congestive failure. Seymour et al.[5] found that in a series of 261 cases of congestive failure culled from various sources, the mean plasma protein concentration was 5.67 grams per 100 cubic centimeters. They suggested that such a decrease in serum proteins might be the result of any one or a combination of three factors: (1) inadequate protein intake; (2) excessive protein loss; (3) failure of protein synthesis. The problem was further complicated by the fact that no one had shown whether the decrease in serum proteins was absolute or relative. That is to say, since the plasma volume is greatly expanded in congestive failure, the low plasma concentration of proteins might simply be due to the failure of protein synthesis to keep pace with the expanding fluid volume. That this is exactly the case was indicated in this experiment by virtue of the fact that the total serum proteins was always higher in chronic congestive heart failure during episodes of decompensation than it was during periods of normalcy in any given patient. In view of these revelations, the decreased

serum protein concentration is not seen to occur as a result of the increase in total body fluids. However, it must be noted that once the formation of edema has already proceeded to the point wherein the blood volume is so inflated that the protein depots can no longer supply it with sufficient serum proteins, then conditions arise which will aggravate the degree of edema already present. The reason for this has been demonstrated by Warren, Merrill, and Stead[6] who found that in the presence of low serum protein values there is a disproportionate increase in the volume of the interstitial fluid compartment as compared to the intravascular fluid compartment. It must be emphasized, therefore that hypoproteinemia is a result and not a cause of cardiac edema, but that when the latter condition has led to its formation, it may then aggravate this condition.

In way of summary we are now enabled to say that the classical concepts regarding the formation of cardiac edema must be abandoned because: (1) it has been shown that increased capillary permeability probably does not exist in the congestive failure syndrome, and that if it does it cannot conceivably contribute appreciably to the formation of edema; (2) increased venous pressure is the result and not the cause of the plethora and edema which characterize the state of congestive failure; (3) decreased serum protein concentration is the result and not the cause of the accumulation of abnormal amounts of fluid in congestive failure.

It is now apparent that we must abandon the "microscopic capillary segment" viewpoint and consider the organism as a whole, with particular reference to fluid balance, if we are to make any progress in the understanding of the true mechanisms leading to cardiac edema. Let us then begin with certain well known facts concerning water balance: (1) all water which is ingested is absorbed from the alimentary tract; (2) a certain variable, yet relatively constant portion of this is excreted via respiration, perspiration, and defecation; (3) a variable quantity of this water, depending upon the needs of the organism, is excreted by the kidney.

The kidney then is the organ which is primarily concerned with the maintenance of a normal volume of fluid within the body. Should this organ fail for some reason and the water continue to be ingested at its former rate, then obviously a state of edema will result. This is very nicely illustrated in the case of patients who suffer acute renal shutdown following hemolysis and blockage of renal tubules. Such patients characteristically develop massive edema.

Let us now examine the state of water balance in congestive failure: (1) the generalized edema must result from either an excessive intake or an inadequate output of fluids. Since oliguria and not polydipsia characterizes the state of congestive failure, then failure of kidney function must be at fault; (2) the kidney of congestive failure exhibits no anatomical lesion which could account for this functional failure. Therefore the answer must be sought in some functional aberration or altered physiology of this organ.

The problem now arises as to how we may relate failure of the heart with failure of the kidney. In attempting to determine the precise pathogenesis of this condition, the dictates of logic command that we begin with the primary seat of the disease, namely the heart. Uncompensated myocardial insufficiency may be defined as the failure of the heart, as a pumping mechanism, to maintain a minute output of sufficient volume to supply the demands of the tissues at a given time. Such a state of affairs is intolerable and cannot long be endured. Reasoning along teleological lines one may assume that the organism will react in some manner either to minimize or offset this disturbance. One way by which such compensation could be effected would be an increase of the diastolic filling of the heart which, in accordance with Starling's law of the heart, would enable this organ to contract with greater vigor. This increased diastolic filling could in turn be achieved if the volume of fluid contained in the vascular tree were to be increased.

This precise sequence of events has been beautifully demonstrated in isolated heart-lung preparations by Katz et al.[15] These investigators, working with dogs' hearts found that after a variable period of normal function, the hearts would begin to manifest signs of failure as determined by the decrease in cardiac output and heart's work. In attempting to simulate the conditions of congestive failure, the peripheral resistance was increased in order to maintain a constant arterial pressure (patients with congestive failure characteristically maintain their blood pressure levels). In spite of these efforts, the hearts continued to fail, the venous pressure showing at no time a tendency to rise. If, however, they increased the circulating fluid volume by injecting fluid into the closed conduits of their apparatus, then there was an immediate marked rise in venous pressure leading to an increased diastolic filling of the heart which would then begin to contract with greater vigor as manifested by a rise in cardiac output and heart's work. This state of affairs would persist for a time until the heart's output would again begin to fall. A further increase of circulating fluid volume would then again restore output to its former level. They were thus enabled to maintain a constant cardiac output by successive increments in the volume of fluid until a point was reached where further stretch of the cardiac fibers did not invoke a beat of increased vigor, whereupon the beats became progressively weaker and rapidly ceased.

Although one is not justified in identifying such experiments, in which all of the physiologic variants in the intact organism cannot be simulated, with the sequence of events in congestive failure, one can not deny that these observations are strongly suggestive.

Now let us return to the picture as it presents itself in man. The only way by which blood volume and with it interstitial fluid volume can be increased is, as indicated earlier in this paper, by means of decreased renal output in face of a normal fluid consumption. This we know is precisely what takes place in the edema-accumulatory phase of congestive failure. In order that an anatomically intact kidney form less urine, one or both of two things must happen: (1) less blood must be delivered to the kidney; (2) more tubular reabsorption of water must take place.

Merrill[16] conducted an experiment designed to determine what abnormality of kidney function led to the oliguria of congestive heart failure. Following the methods devised by Smith and co-workers, he determined renal blood flow and filtration rate using the p-amino hippuric acid and inulin clearance technics on a series of normotensive cardiac patients without obvious renal disease. Patients were selected who would tend to become edematous at bed rest when mercurial diuretics were withheld so that measurements could be made at a time of increasing decompensation. At the same time as the renal function studies were carried out, the cardiac output was determined by the direct Fick principle, using radio-opaque ureteral catheters passed from the cubital vein to the right atrium under fluoroscopic control, this being the most accurate known method of determining cardiac output. He found that in patients with congestive failure renal blood flow was reduced to one-third to one-fifth of normal. The filtration rates were from one-half to one-third normal, giving filtration fractions from 30 to 50 per cent as contrasted with normal values of 20 per cent. It was further found that renal blood flow was reduced to about one-fifth normal when cardiac output was approximately one-half normal, indicating a specific diversion of blood away from the kidney. In the whole series there was a high degree of correlation between the cardiac index (cardiac output per square meter of body surface area) and renal blood flow. This experiment demonstrates in a most straightforward and unequivocal manner that the decreased urinary output in congestive failure is correlated with and dependent upon a reduced cardiac output. Proponents of the "backward theory" of failure might raise the objection at this point that the reduced renal

blood flow might have resulted from the high venous pressure causing congestion in swollen kidneys contained in a tight capsule. This objection was very clearly overruled by the investigator with several patients in whom venous pressure was reduced to normal with mercurial diuretics and kept at a normal value for several days. The renal blood flow still persisted at its low values, demonstrating that venous congestion did not cause this reduction. One of the most interesting facts demonstrated in this experiment is that whereas in this series of patients the cardiac output was rarely reduced below one-half the normal resting value, the renal blood flow was frequently reduced to approximately one-fifth normal, indicating a specific diversion of blood away from the kidneys, organs which normally receive about 20 per cent of the cardiac output.

Although Merrill did not discuss the problem further, it is of some consequence to relate this specific diversion of the blood from the kidney with known facts concerning renal hemodynamics. The vascular architecture of the kidney differs markedly from that of any other organ in the body because of the fact that blood in its course through this organ must pass through two sets of arterioles and two sets of capillary networks.

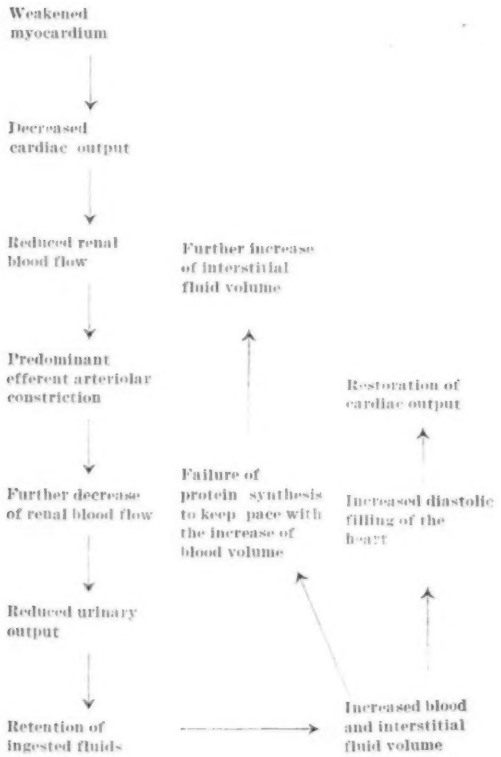
The degree of resistance to blood flow through an organ is determined largely by the degree of constriction of its arterioles. If the kidney arterioles were to be constricted to a greater degree than those of other organs in the body, then it is manifestly clear that blood will be proportionately shunted away from the kidney. The question now arises as to why there should be a greater constriction of kidney arterioles, and if so, which set is constricted. Smith[17] answered these questions by demonstrating that the degree of constriction of the efferent arteriole is delicately attuned to variations in renal blood flow. When renal blood flow falls, this arteriole constricts so that intraglomerular pressure, which is related to the relative state of constriction of the afferent and efferent arterioles, is elevated. This independent efferent arteriolar constriction operates so that within wide limits of renal blood flow, a remarkably constant rate of filtration is maintained. Now the higher the intraglomerular pressure, the greater will be the amount of fluid filtered from a unit volume of blood. The percentage of fluid filtered from a unit volume of blood is designated as the filtration fraction and is defined by the ratio: glomerular filtration in c.c. per minute divided by the renal blood flow in c.c. per minute. When this is elevated, it signifies predominant efferent arteriolar constriction. The filtration fractions of the cardiac patients in Merrill's experiment were consistently from one and one-half to two and one-half times the normal values. This then affords the obvious answer for the question why renal blood flow to the kidney was so markedly reduced.

It is of interest to note that Phillips et al.[18] found that a similar diversion of blood flow away from the kidney occurred during experimentally produced hemorrhagic and traumatic shock in dogs. The basic circulatory derangement in both cardiac failure and shock is inadequate output of the heart. In shock it is inadequate venous return which affects cardiac output adversely; in congestive failure it is intrinsic weakness of the myocarium.

On the basis of firm experimental evidence, we are now enabled to trace a remarkable sequence of events whereby urinary flow is diminished in answer to the demands of a weakened myocardium for a greater blood volume with which its diastolic filling will be increased.

The symptomatology of the congestive failure follows logically from such a sequence of events. It has already been mentioned that the increased venous pressure is due to the overfilling and distention of the vascular system. Gibson and Evans[4] lend further support to this with their observation that the average degree of rise in blood volume parallels the average degree of rise in venous pressure in patients with chronic progessive congestive failure.

The reason for the common association


Weakened myocardium
Decreased cardiac output
Reduced renal blood flow
Predominant efferent arteriolar constriction
Further decrease of renal blood flow
Reduced urinary output
Retention of ingested fluids
Further increase of interstitial fluid volume
Failure of protein synthesis to keep pace with the increase of blood volume
Increased blood and interstitial fluid volume
Restoration of cardiac output
Increased diastolic filling of the heart

of an abrupt rise in venous pressure and the first signs of clinically apparent edema probably lies in the fact that the elastic limit of the confining skin membrane is being approached. This will be accompanied by an abrupt rise in tissue pressure which will in turn be simultaneously reflected by an upward deflection in the venous pressure.

Although the relationship between a failing myocardium and a weakened vigor of the heart's beat and consequently a drop in cardiac output would seem to be such an obvious sequence of events that the question of its presence would require no defense; such, unfortunately, is not the case. Cardiac output has been investigated extensively by many workers using a variety of technics. The results on the whole have been very contradictory. An exhaustive review of the various procedures with an attempt to evaluate their validity is beyond the scope of this paper. However it is felt that a discussion of some of the broader aspects of the problem may prove helpful.

Harrison,[10] in an extensive survey of the literature, found that the great majority of investigators agree that the output per minute of the heart is subnormal in patients with cardiac diseaese. On the other hand "repeated observations on the same individual in dicate that clinical improvement with the disappearance of congestive manifestations may be associated with an increase, a decrease, or with no change in the cardiac output per unit of time." This has been considered to be strong evidence against any theory of congestive failure based upon a reduction of cardiac output.

Those who raise these objections must fail to realize that as indicated in the scheme presented above, as soon as there is a momentary fall of cardiac output, compensatory mechanisms go into play to restore volume flow. Hence in a patient with a failing myocardium who is ingesting average quantities of salt and water, blood volume will increase by a given increment with each episode of failure, so that a patient may gradually lapse into a severe congestive state without showing at any given time a significant diminution of cardiac output. With regard to the objection that a given individual in severe failure may have a higher output than at a time when his condition is improved, it must be pointed out that the output of the dyspneic, acutely ill patient in severe congestive failure can by no stretch of the imagination be considered a basal output. No valid conclusions can be drawn from a comparison of outputs which are not strictly basal because exercise alone may drive cardiac outputs to heights tenfold above normal. With these considerations in mind, one must feel that no one can seriously challenge the concept that the cardiac output tends to drop in myocardial insufficiency.

Much attention has been centered in recent years upon the role of the sodium ion in the pathogenesis of cardiac edema. The work of Burch and Reaser,[1] Warren and Stead,[2] Schemm[19] and others has established beyond question the extremely important

part played by the sodium ion in the formation of cardiac edema. No theory regarding the pathogenesis of this condition can be considered either complete or acceptable unless it can account for the edema promoting effect of this ion. One can most logically approach this question by considering what possible effects the sodium ion may have upon the modus operandi of the two organs most vitally concerned in the congestive failure syndrome, namely, the heart and kidneys.

Let us begin with a consideration of the heart. It is known from perfusion experiments that the sodium ion is an essential constituent of the perfusate. Hence a deficiency of this ion might conceivably exert a depressant effect upon the myocardium. However, this is of no help because it is the ingestion of salts, not their absence which tends to produce edema. Furthermore no serious chemical imbalance of the composition of body fluids with respect to sodium seems to exist in congestive failure patients. And finally the sodium ion exerts an aggravating effect upon other types of edema which are not associated with cardiac derangement. Hence we may assume with reasonable assurance that the effect of sodium is not mediated through the heart.

Let us now consider the effect upon the sodium ion of function of the kidney. Earlier in this paper it has been shown how this organ is concerned with the maintenance of the volume of body fluids, and it has been indicated that this is controlled by hemodynamic mechanisms regulating the volume of renal blood flow. For the sake of brevity and simplicity, the tacit assumption was made throughout this discussion that the sodium content of ingested fluids was isotonic with the body fluids. However, in reality this is rarely if ever the case, and whenever an imbalance exists the kidney reacts in such a manner that the ionic concentration of the body fluids is maintained at a remarkably constant level. This ability of the kidney to maintain the constancy of the electrolytic pattern of body fluids is primarily the function of the tubules. The precise nature of these processes has been the subject of intensive investigation, a discussion of which would lead us far afield into the realm of renal physiology. However certain relationships have been clearly demonstrated and are pertinent to this discussion.

When water is taken into the body without sodium, the body fluids are diluted. This presumably prompts the liberation of a hormone which depresses the tubular reabsorption of water. Hence a more dilute urine is excreted until a normal body fluid concentration is attained. On the other hand, when sodium without water is taken into the body, the fluids become concentrated with respect to sodium. This presumably depresses the formation of the diuretic hormone, more water being reabsorbed through the tubule, so that a more concentrated urine is excreted until the normal electrolytic pattern is again attained.

It is then obvious that circumstances will arise in which the kidney, in the defense of the *composition* of body fluids, will have to sacrifice its defense of the *volume* of body fluids; or conversely, it may have to sacrifice composition for volume.

For example, let us consider the case of a patient in progressive congestive failure. If we were to supply this patient water and at the same time withhold sodium, the following sequence of events can be envisioned as taking place: (1) because of decreased renal blood flow due to low cardiac output, this water will tend to be retained leading to a *decreased* urinary excretion; (2) as a result of this retention, the body fluids will be diluted; (3) as a result of this dilution, the tubular reabsorption of water will be repressed leading to *increased* urinary excretion. The net effect upon the volume of urine excreted will then be the resultant of these two opposing mechanisms, the decreased filtration tending to decrease urine volume and the decreased reabsorption tending to increase urine volume. As has been pointed out by Peters,[20] the preservation of normal electrolytic pattern seems to be the most fundamental concern of the kidney. Therefore, in the example cited above, decreased tubular reabsorption will

exert a predominant effect so that any ingested sodium-free water will be promptly excreted. This is in accord with the clinical observations of Schemm[19] who found that on low salts diets patients with chronic congestive failure did not retain water even though they consumed large quantities.

One is then led to suspect that the cardiac patient who is on a normal diet must be consuming a quantity of water which when added to his salt intake will yield a hypotonic solution. He will then excrete that portion of his water intake which is above the amount required to combine isotonically with his ingested salt. If we now increase his salt intake, he will retain a much greater part of his water intake thereby greatly facilitating the rate of edema formation. That the average diet is in reality hypotonic can easily be seen from the fact that the average sodium ingestion of ten grams when mixed with the average total water intake of two liters yields a 0.5 per cent solution. Because of the fact that a certain proportion of the sodium is obligatorily excreted in combination with acid metabolites, the amount of sodium available for storage in the body fluids is even less than indicated above.

The foregoing explanation for the edema-promoting effect of the sodium ion and the salutary effects which are seen on low salt diets is in complete conformity with the mechanism for cardiac edema proposed in this paper.

THERAPEUTIC IMPLICATIONS

Such a radical departure from the conventional concepts regarding the nature of congestive failure as has been set forth in this paper demands a re-evaluation of the therapeutic measures in vogue. Since the accumulation of excess fluid volume is compensatory to the failing heart, the question will be raised as to why such beneficial effects are observed following measures designed to reduce this volume. The answer to this probably is as follows: All patients with chronic congestive failure except in the most extreme terminal stages retain some degree of cardiac reserve. When these patients, in the course of whatever activity they may participate in become decompensated, they are put to bed. While thus confined, their resting cardiac output can be maintained at a lower blood volume than could the larger output demanded by even moderate activity. Therefore with bed rest alone these patients should begin to lose large quantities of fluid. If one gives digitalis, the heart's action is further improved and an even greater diuresis will result. The action of diuretics is probably simply to catalyze the mobilization of fluids which would take place in time without drugs. It must also be realized that the symptoms of which the cardiac complains most bitterly are those consequent to the plethora and edema which greatly embarrass his respiratory and other physiologic functions. He may feel better in a state of mild shock due to inadequate output than he does with adequate output and severe dyspnea and yet be closer to death. However, dyspnea itself will increase the disproportion between cardiac output and body demands, and should be alleviated as promptly as possible.

One cannot however escape the conclusion that too rigorous salt restriction might deprive an occasional patient of the blood volume he requires, even at resting levels, to prevent him from making a premature exit in shock. One also wonders if the sudden collapse and death of a patient following massive mercurial diuresis might not be explainable upon this same basis.

SUMMARY

1. The classical concepts regarding the causation of cardiac edema have been reviewed and found untenable in the light of recent experimental observations.

2. Experimental evidence pointing toward a new concept of the mechanisms involved in the condition have been cited.

3. On the basis of this experimental evidence combined with a teleological approach a hypothesis has been constructed which would appear to explain some of the puzzling aspects of the problem of cardiac edema.

4. Some therapeutic implications of this hypothesis have been discussed.

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HYPERTROPHIC PYLORIC STENOSIS*

H. O. BARKER, M. D.†
H. H. HARDY, JR., M. D.†
AND
H. A. WHITE, M. D.†
ALEXANDRIA, LA.

Hypertrophic pyloric stenosis is one of the rare anomalies of development of the stomach. The condition has its onset between the second and tenth week and is characterized by five major symptoms: (1) vomiting, which is projectile in type and frequently repeated; (2) strong peristaltic waves, plainly visible on the epigastrium, and always moving from left to right; (3) loss of weight, which is often alarming; (4) infrequent and scanty stools, containing mostly mucus and very little fecal matter as seen in "starvation stools", and (5) by a hard, oval tumor, representing the hypertrophic pylorus. The tumor cannot always be palpated and is frequently missed.

PATHOGENESIS

The pathogenesis of this condition continues to be a subject of dispute. Some physicians feel that the condition is due to spasm of the circular fibers of the pylorus which leads to hypertrophy due to the increased muscular activity, while others hold to the theory of a primary or congenital hypertrophy with a superimposed spasm. The latter theory has the greatest number of adherents. The pathology consists of hyperplasia and hypertrophy of the circular muscle of the pylorus. The pylorus is "olive-shaped", and twice the size of a normal pylorus and is sharply demarcated from the rest of the stomach and duodenum and is nearly as firm as cartilage. The stomach is usually, but not always dilated, and the muscular wall is thickened, especially at the pyloric end.

CLINICAL PICTURE

The clinical picture is dominated by the five major symptoms as mentioned previously. Vomiting is the initial symptom. It usually starts gradually between the second and tenth week and becomes progressively more severe and constant. Very soon every feeding is vomited and quite frequently a feeding is vomited before it is finished. Vomiting interferes with the infant's nutrition and is followed by a severe loss in weight, amounting to several ounces a day. The character of the stools changes as the amount of food reaching the intestines becomes smaller; finally, the starvation stools contain mostly mucus and very little fecal matter. Peristaltic waves travel-

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†From the Baptist Hospital, Alexandria, La.

ing left to right on the epigastrium can be seen in all cases and are most pronounced while or just after the baby has taken a bottle. With the baby placed between the examiner and the light, there appears a mound-like prominence medial to the left costal arch which travels toward the right. Then follows a succession of such prominences separated by depressions or troughs, moving slowly toward the right and disappearing under the right costal arch. There are remissions in the peristaltic waves—then suddenly they will reappear, and when they are most active, the infant will be in evident distress and will squirm and cry and frequently vomit projectilly.

The pyloric tumor can at times be palpated, but is more often not palpable.

Simple pylorospasm may be confused with hypertrophic pyloric stenosis, but in these cases it is less severe. The vomiting is milder—there is no loss in weight, but usually a slow, steady gain and the stools are not starvation in type, in that they contain very little mucus.

Due to the persistent vomiting with the loss of acid, there is at times an alkalosis present and in patients who do not go to operation relatively soon, there exist shallow respirations with a diminishing rate to as low as six to eight per minute with well marked periods of apnea.

The roentgen findings in congenital pyloric stenosis are of great value and are usually decisive.

TECHNIC

A small catheter is passed into the stomach through the nares if not too narrow, but if they are too narrow, the baby will swallow the catheter easily because it is always hungry. The catheter is followed with the fluoroscope to be sure it does not enter the lungs. If there is a great deal of air in the stomach, some of it may be withdrawn. This accomplished, place two ounces or 60 c.c. of barium mixture into the stomach with a large syringe or by gravity. Films are made in the supine and prone positions every two hours for six to eight hours. If the stomach is not empty, another film is made in 20 to 24 hours the next morning, and barium will usually be present in the stomach if there is stenosis. The important roentgen signs of pyloric stenosis are elongation of the pyloric canal, delay in or failure of opening of the pylorus, prolonged gastric emptying time, and gastric dilatation with or without excessive peristaltic activity. The base of the duodenal bulb is usually indented. The most informative film to show hypertrophy of gastric musculature is obtained with the patient in the supine position with the left side slightly raised. If there is gas in the transverse colon, the thickness of the gastric wall can be fairly accurately measured from the thin line of the transverse colon to the barium line in the stomach. If the soft tissue shadow measures 4 mm., then hypertrophy should be considered likely, and if over 5 mm., it is certain. In this position the mass may also be felt by the roentgenologist. A large gas filled stomach with very little gas beyond the stomach is also helpful in making the diagnosis. Pylorospasm and hypertrophic pyloric stenosis are differentiated chiefly by the presence of a mass and by the more constant and more marked clinical manifestations, and roentgen signs of obstruction in the latter.

The care of an infant with hypertrophic pyloric stenosis presents a problem that requires careful and adequate therapy directed both by the pediatrician and attending surgeon. Immediate hospitalization is mandatory for this regime in order that careful appraisals of the results of the treatment may be made. In those infants who present a picture of still a fair to good state of nutrition and hydration and who have shown evidences of the disease for only a short time non-operative therapy is the procedure of choice as long as the patient improves and shows no signs of increasing symptomatology. This type of treatment consists of antispasmodics, thick feedings and sedatives with fluids supplemented as required. We believe and are convinced from the care of these patients on our service that any infant who shows extreme de-

hydration, a progression of the symptoms, a continued weight loss or who in any respect fails to respond within 24 to 48 hours of non-operative treatment should be operated upon. In addition, we believe that any infant with this condition who shows signs and symptoms that are severe on admission or any patient whom the history shows to have had the disease over a period of say one week or more should be operated upon.

We consider each of these cases an emergency and once the diagnosis is established all the patients are considered potential candidates for operation and they are immediately treated with that idea in view. As we see more of these patients and the excellent results from operation, we believe less and less in the non-operative treatment and are now proceeding almost entirely with surgical therapy. As soon as the diagnosis is confirmed, these patients are operated upon, the only preoperative requirement being that the infant be in a state of normal hydration, the red cell and hemoglobin be in normal range and the general condition of the patient be such that an operation can be done with a good chance of survival. It cannot be too strongly emphasized that the preoperative care of these infants is the determining factor in their postoperative recovery.

COMPLICATIONS

The surgical procedure is the now recognized and established Fredet-Rammstedt operation which is a standardized procedure and differs only very little in its routine applications. We use routinely local anesthesia supplemented by some type of mild preoperative sedation, believing that general anesthesia increases the mortality and postoperative complications. Both the transverse and right rectus incisions have been employed and we prefer the right rectus. The transverse incision does, however, have the advantage of better exposure in those cases in which the pylorus is hard to deliver because of its short attachments and reduced mobility. The only complications of importance during the operation are perforation of the mucosa and hemorrhage. Perforation occurred twice in our series, once on the duodenal and once on the gastric side. The rent can be closed by suture followed by omental grafts or suturing of the omentum over the perforation or a second parallel incision may be made through the muscle and the tissue shifted over to cover the rent, it being held in place with sutures. Hemorrhage is at times annoying and originates from the small vessels in the pyloric area. It can be controlled by ligatures or sutures or at times by simple pressure. More recently we have employed the use of thrombin topically in these cases and have found it to be a rapid and simple means of controlling this annoying difficulty. These patients are given fluids by mouth beginning about four to six hours post-operatively and gradually returned to their normal feedings. Supplementary fluids are usually given subcutaneously as required.

CONCLUSION

Under this plan of treatment, we (H. H. Hardy) have operated upon 18 of these patients and all have recovered normally. There have been no postoperative complications in the series and no deaths.

DISCUSSION

Dr. H. A. White (Alexandria): I have enjoyed Dr. Barker's paper very much; it's a very excellent paper, and, as he told you, I had something to do with writing it.

I would like to stress one point: The collaboration between the surgeon and the pediatrician, or the physician, in these cases. I think our success in Alexandria has been due to the fact we have not let these patients go too far with pyloric stenosis before they were operated upon.

If they become moribund or are almost moribund, then your risk of saving a patient's life is very much greater.

I would like to stress as cardinal signs those that Dr. Barker mentioned, because they are very important, and in that lies your diagnosis. The projectile vomiting is the most frequent. The peristaltic waves, which always pass from left to right; you can see three mounds, and two depressions between those mounds are moving from left to right. The palpable pyloric tumor is present. I must admit, maybe my fingers are not acute enough, but I have never felt one. When it is attached very deep, the pyloric incision is quite difficult to palpate, although it looks like it can be palpated.

Some babies rock along three or four days; if they do not gain and all these symptoms begin,

then that more or less is in keeping with the diagnosis.

Constipation, as he mentioned, is always present, going into, after a few days, what we call a constipated stool. There is another symptom he did not mention, which is abdomen pain. I think these infants have pain, particularly after taking the bottle. They are very irritable and cry a lot. I believe there is very definite pain.

There is a question of the pathogenesis of this situation. I think it is a congenital anomaly: a hypertrophy of the pylorus and followed by the spasms of the muscles which occur, as he told you, around two to six weeks.

We had one case some years ago in a newborn. It was very definite from the start. The child vomited immediately, as soon as they began to feed it. There was constipation; there was subjective vomiting, abdominal pain. We operated on this child on the sixth day, with a typical pyloric stenosis, and the child had an uneventful recovery.

We had another case some years ago that was very interesting, in that it had two situations existing: a stenosis of the esophagus of the cardiac portion and also the pylorus. It happened not to be a hypertrophic affair. I would like to read you the x-ray report we had on that, at this time:

"The lower end of the esophagus shows a stenosis with dilatation above and retention of barium in the dilatation. The barium, however, passes into the stomach, which is filled with gas and after rotating the patient to the right, the barium passes into the duodenum which also shows a restriction, or stenosis. In three hours considerable barium had emptied out of the stomach. A 24 hour film shows a faint outline of the dilated esophagus with constricted cardiac portion and also constriction in the pyloris ring. There is a slight 24 hour retention in the stomach but much less gas as a stomach tube had been used. The barium had passed to the sigmoid. An examination later in the afternoon of the same day showed the stomach empty and the barium in the rectum. An enema resulted in the evacuation of some of the barium.

"The impression was congenital stenosis of the esophagus and the pylorus.

"A recheck of this infant six days later—recheck of the stomach shows a constriction at the lower end of the esophagus with accumulation of barium in the esophagus. However, the barium passes more rapidly than on the previous film. The barium also passes somewhat more rapidly through the pylorus."

This infant had probably more of a spasm than it had a stenosis; even probably more than a stenotic condition of the pylorus. We were successful in passing a small tube into the stomach and, as I remember, we increased the size. Without it, it was a question of passing it through an opening somewhat restricted. The child made an uneventful recovery in 20 days. There was vomiting, but not the projectile type: his stomach would fill up and he would spit it right back, immediately. And he kept on like that.

So, this is a case where we had two anomalies which, incidentally, turned out very well. This child now, by the way, is 15 years old and doing very nicely.

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THE TREATMENT OF CHRONIC ANEMIA*

NORTON W. VOORHIES, M. D.

NEW ORLEANS

An anemia is an abnormal clinical state, which can be treated successfully only by a thorough understanding of its cause. The causes of this condition are numerous, and oftentimes can be determined only by considerable clinical and laboratory study.

The presence of an anemia requires, as do all other medical conditions, a careful history and physical examination. The history of jaundice in the family, or of bleeding from the gastrointestinal tract in a patient, naturally helps to orientate the examiner's thinking and leads to a correct diagnosis. The physical signs of an enlarged liver or spleen, or the presence of pallor, or superficial glandular enlargement, likewise aid materially in arriving at a correct conclusion even before blood studies have been completed.

While a simple blood count is often sufficient, it is advisable to have other determinations, such as a hematocrit, mean corpuscular volume, and mean corpuscular hemoglobin concentration. Through the use of the latter procedures, the recognition of various types of anemia, based on cell size and their hemoglobin content, is made easier. Oftentimes, there is a need for gastric analysis, and gastrointestinal x-rays. Bone marrow studies are in many instances invaluable, though they do require the services of someone trained in their interpretation. The icterus index and the fra-

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gility test may be a necessity, and can be done easily in any laboratory. Special procedures, such as tests for sickling and cold hemolysins, require no unusual skill and only ordinary equipment.

Reznikoff,[1] in an article on chronic anemia, submitted the following useful classification based on etiology, which is listed below with minor changes:

I. Deficient bone marrow function.
  A. Faulty bone marrow nutrition.
    1. Iron deficiency.
      a. Chronic blood loss.
      b. Defective iron ingestion or absorption.
      c. Pregnancy.
    2. Extrinsic or intrinsic factor deficiency.
    3. Protein deficiency.
    4. Thyroid deficiency.
    5. Copper and other heavy metal deficiencies.
  B. Depression of bone marrow.
    1. Toxic nephritis.
    2. Chemical (benzol, gold, sulfonamides, arsenic).
    3. Physical (x-ray, radium).
    4. Chronic infection.
    5. Mechanical, such as leukemia, carcinoma, Hodgkin's disease.
    6. Refractory, such as aplastic anemia.
II. Hemolytic.
  A. Intrinsic.
    1. Hemolytic spherocytosis.
    2. Sickle cell anemia.
    3. Mediterranean anemia.
    4. Paroxysmal nocturnal hemoglobinuria.
    5. March hemoglobinuria.
  B. Extrinsic.
    1. Malaria.
    2. Hemolytic — Streptococcus and staphylococcus infections.
    3. Sulfonamides.
    4. Lead.
    5. Favism.

THE MICROCYTIC ANEMIAS

The iron deficient anemias are characterized by red blood cells which are small and poorly filled with hemoglobin, and thus produce a microcytic hypochromic anemia. The existence of this type of anemia is quantitatively shown by a low mean corpuscular volume, mean corpuscular hemoglobin, and mean corpuscular hemoglobin concentration, the latter usually below 30 per cent. The bone marrow is normoblastic.

An iron deficiency anemia is produced by chronic blood loss and defective iron ingestion or absorption, and by pregnancy. Chronic blood loss is the most common cause of anemia and may be due to a host of things, including bleeding hemorrhoids, esophageal varices, polyps in the bladder or colon, a duodenal ulcer, or hookworm infestation. An iron deficient diet will produce an anemia in infants and children. Iron absorption is interfered with in gastrointestinal disorders, such as chronic diarrhea, gastric resection and anastomoses, and diaphragmatic hernia. Murphy and Hay[2] report an incidence of 26.6 per cent of iron deficiency anemia in the latter condition. A microcytic anemia occurs in pregnant women on an iron-deficient diet because of the demands of the growing fetus. In some microcytic anemias there is a combination of factors, for example, blood loss through menstruation and a deficient diet, as in chlorosis; chronic blood loss, iron deficient diet, and familial predisposition, as in idiopathic hypochromic anemia of middle aged women.

The symptoms of anemia are many. The symptoms common to all types are weakness, palpitation, and rapid pulse. When anemia is severe, ankle edema may occur. There are symptoms and signs characteristic of certain types of microcytic anemias, for example, sore tongue, achlorhydria, diarrhea, dysphagia, and koilonychia, as seen in idiopathic hypochromic anemia. Of special interest is the fact that 39 per cent of patients with chronic hypochromic anemia have some papillary atrophy of the tongue, but this is usually not so severe as that seen in pernicious anemia.[3] According to Darby,[4] chronic iron deficiency alone can produce glossitis and angular fissures. He reports six cases in which improvement

occurred only after the addition of iron to the diet.

The routine use of preparations containing small amounts of iron, liver extract, and vitamins is to be condemned, as usually no one substance is present in a sufficient quantity to relieve the condition should a deficiency in any one of these substances exist. This practice often results in a therapeutic failure, a loss of time, and an unnecessary expense to the patient.

The treatment of the iron deficient anemias is most satisfactory, and, at times, dramatic. An increase in the percentage of reticulocytes follows the administration of iron, and unlike pernicious anemia, the magnitude of response is related to the amount of hemoglobin. The maximum response usually occurs in five to ten days after the beginning of therapy. According to the studies of Balfour et al.,[5] the mucosa of the intestinal tract will absorb iron only when there is a depletion of iron stores in the body. Iron balance is controlled by absorption rather than excretion, which is negligible. Iron given to an anemia not caused by iron deficiency is without effect.

It is of primary importance that bleeding be stopped whenever possible. This is usually not difficult in patients with hemorrhoids and menstrual disturbances. However, it may be a serious problem in a duodenal ulcer or esophagel varices.

It has been shown that ferrous iron is more completely absorbed than ferric iron. Consequently, the former can be used in smaller dosage with as good a result and with less gastrointestinal irritation. There seems to be no justification for the use of the ferric salts. The ferrous salts used today are: Ferrous sulfate, 15 grains per day; ferrous gluconate, the same dosage; and ferrous carbonate, 12 grains daily. Molybdenized ferrous sulphate has been used in the treatment of iron deficiency anemias. Neary[6] reports a more rapid response in the microcytic hypochromic anemias of pregnancy with this drug than with plain ferrous sulfate. According to his report, gastrointestinal disturbances are less frequent. An effective iron preparation will increase the hemoglobin 1 to 2 per cent per day and will produce a reticulocyte response. It is best to give iron preparations after a meal, as the drug is better tolerated at this time. It is also advisable to start with a small dose, for example, one 5 grain tablet daily for several days, then two tablets for several days, then finally one after each meal. This method of treatment helps to prevent annoying gastrointestinal symptoms, such as nausea, vague abdominal discomfort, and occasional diarrhea. Iron is so effective by mouth that there is no justification to give it parenterally. Intravenous iron in therapeutic dosage is expensive and painful, and, at times, dangerous. Goetsch, Moore, and Minnich[7] gave large doses intravenously, and though a good therapeutic result was noted, there was a high incidence of toxic reactions, such as nausea, vomiting, abdominal pain, and diarrhea. Since copper is present as a contaminant in most iron preparations, and as it is present in the normal diet, the addition of copper is not necessary in the treatment of the iron deficiency anemias of adults. There are those who believe that the microcytic anemia of infants requires copper. However, this is a controversial point. In addition to iron, an adequate diet is important. The diet should include liver, red meat, and eggs. Supplementary vitamins are not necessary unless a definite deficiency exists.

THE MACROCYTIC ANEMIAS

The macrocytic anemias are those anemias which are characterized by an abundance of large cells, macrocytes, which are adequately filled with hemoglobin. In such cases, the mean corpuscular volume and the mean corpuscular hemoglobin are increased. The mean corpuscular hemoglobin concentration is usually not altered. The bone marrow is megaloblastic in type. A macrocytic hyperchromic anemia is found in pernicious anemia, and may occur in sprue, tropical and non-tropical, the nutritional anemias, the anemia of pregnancy, and of liver cirrhosis, myxedema, *Diphyllobothrium latum* infes-

tations, and following gastrectomy and intestinal anastomoses. While a macrocytic hyperchromic anemia usually occurs in sprue, there are some cases in which the blood picture is microcytic or normocytic in type. The same applies to idiopathic steatorrhea, or non-tropical sprue, the anemia of pregnancy and of liver cirrhosis. More often than not, a microcytic hypochromic anemia follows gastrectomy and intestinal anastomoses. The pernicious anemia of fish tapeworm infestation is rare. It has been estimated to occur in only 0.1 to 0.2 per cent of persons harboring the parasite. There are those who believe that pernicious anemia in this condition is coincidental. A mild macrocytic anemia occurs in myxedema and seems to respond only to thyroid and not to liver extract.

The macrocytic anemias are treated with some form of liver extract, with the possible exception of that found in myxedema. In pernicious anemia there is an absence of the erythrocyte maturation factor. According to the hypothesis of Castle, the intrinsic factor is not present in the stomach juices to combine with the extrinsic factor in food. Consequently, maturation of the red blood cells is arrested at the megaloblastic stage. Liver and liver extract can be given by mouth, and though effective, are not practicable, as seldom can sufficient quantities be taken orally to restore the blood picture to normal. Liver extract intramuscularly is the route of choice. The average case should be given 15 units daily for one week, and then 15 units twice a week until the red blood cell count and the mean corpuscular volume have reached normal levels. When there are no neurologic complications, injections at two or three week intervals are sufficient, estimating the dose so that the patient will get one unit per day. If neurological complications are present, it is advisable to continue weekly injections of liver extract indefinitely, and to use larger doses than are ordinarily used, that is, 2 c.c. of a concentrate, or a total of 30 units per week. This should be supplemented with oral liver, about a pound per week, as the factor present in liver, responsible for the improvement of nervous system lesions, is not known, and this factor could have been destroyed in the preparation of a refined product. Adequate liver therapy arrests the progress of nervous system complications, and in 50 to 60 per cent of cases improvement will occur, provided these complications have not been present too long. Lesions of the lateral columns are most resistant to treatment, while posterior columns, peripheral nerve and sphincter control respond more readily.[8] An improvement in general strength and motor performance can be expected. The presence of an infection or of extensive arteriosclerosis renders liver therapy less effective, and should be looked for whenever the response is poor. The error in liver therapy is usually giving too little rather than too much. Large amounts of liver are stored and are not harmful. The patient with pernicious anemia will require treatment for the remainder of his life.

Recent studies with folic acid indicate that a remission can be produced by its use in pernicious anemia, as well as in other macrocytic anemias.[9] While the drug has not yet had sufficient clinical trial, it seems to be capable of relieving many of the symptoms of pernicious anemia, and of restoring the blood picture to normal or near normal levels. However, its ability to maintain a remission is still an uncertainty. So far as is known at the present time, it has not been effective in preventing or relieving the neurologic relapse in some cases of pernicious anemia.[10] Meyer,[11] in a recent report of eleven cases of pernicious anemia and one of sprue, concluded that folic acid, 15-50 milligrams, given orally, or 20 milligrams given intramuscularly, usually produced only a submaximal reticulocytosis, and failed to prevent the progression of neurologic lesions. However, he found that when folic acid was combined with a small dose of liver extract, one-half unit per day, a reticulocyte response occurred which was greater than when adequate amounts of liver were used alone. There was also im-

provement in the symptoms and signs of combined sclerosis in three patients.

In conclusion, it can be said that the treatment of pernicious anemia with folic acid is still in the experimental stage and that it would be unwise to treat a patient with this drug alone over any prolonged period of time.

The patients with pernicious anemia should have an adequate diet, including liver, red meat, and eggs. Sufficient rest and exercise are also important. Dilute hydrochloric acid is only needed when the patient is having gastrointestinal symptoms due to achlorhydria. In such instances, 4 c.c. of dilute hydrochloric acid (USP) in water or fruit juices may be given before meals. Iron is seldom needed in pernicious anemia, unless there has been a recent blood loss. Rarely is the blood picture of pernicious anemia influenced by an iron deficiency. In such cases, the administration of iron is followed by a more characteristic cell appearance.

The anemia of sprue and idiopathic steatorrhea (non-tropical sprue) is thought to be due to absence of extrinsic factor in the diet. While this anemia is usually macrocytic, it may be normocytic or microcytic in type. These cases are greatly benefitted by liver extract, even those with a normocytic or microcytic blood picture, although doses of iron may be needed as well. It is generally conceded that larger doses of liver extract are needed than are used in pernicious anemia, and the results are not nearly as satisfactory.

In sprue and idiopathic steatorrhea careful attention must be given to diet. though the advent of liver therapy has made diet restrictions less rigid. It has been found that these patients do best on a high protein and low carbohydrate and low fat diet. Calcium may be needed as the deficiency is not uncommon, particularly in non-tropical sprue. Additional vitamins may likewise be indicated. Celiac disease, an equivalent of non-tropical sprue, occurring in children, is usually normocytic or microcytic in type, but this likewise is benefitted by liver extract.

Folic acid, parenterally, or given by mouth, has been found to be effective in sprue, both tropical and non-tropical.[9, 16] The symptoms of diarrhea and glossitis are promptly relieved and blood regeneration takes place. The macrocytic hyperchromic anemia of pregnancy, liver cirrhosis, gastrectomy, and intestinal anastomoses responds to injections of liver extract. Folic acid has been used in the pernicious anemia of pregnancy, liver cirrhosis, and nutritional macrocytic anemias with good results.[9] The macrocytic anemia of fish tapeworm infestation responds to injections of liver extract.

As a result of the observations of Lucy Wills in India, macrocytic anemias occurring in natives of the tropics have been recognized. Most of the cases have occurred in pregnant women. However, cases have occurred in non-pregnant women, and in men as well. Many of these patients failed to respond to refined liver extract, but responded to crude forms of the extract. Watson and Castle[12] reported three cases of macrocytic anemia occurring in pregnancy, which responded only to oral liver extract. 1 unit per day, or 15 c.c., t.i.d., of liquid extract of liver (USP Valentine). A fourth case responded only to intravenous liver in large amounts. Apparently, there are macrocytic anemias which are relieved by some unknown substance in liver which is different from the erythrocytic maturation factor effective in pernicious anemia.

Anemia due to an inadequate diet of protein does occur. Whipple's[13] work on dogs has shown that animals depleted of blood plasma will not regain previous hemoglobin levels until protein is restored, despite large amounts of iron. While such an anemia is common, adequate protein intake may be a factor in patients chronically ill, particularly those with disturbances of digestion due to short-circuiting operations on the gastrointestinal tract.

Those anemias which are due to a depression of bone marrow are relieved only by removal of the depressing agent, which in many instances is impossible. The bone marrow, when depressed by chemicals such

as the sulfonamides and gold, or by physical agents such as x-ray and radium, will recover when the depressing agents are withdrawn. Studies by Greenberg et al[14] on patients with infections have shown that iron intravenously fails to produce an increase in hemoglobin. The excess iron is stored in the liver and the hypoferemia persists. The anemia is thought to be due to retardation of hemoglobin synthesis whereby red blood cells break down after their normal life span and replacements are slow in forming. Mechanical interferences with red blood cell formation, such as are found in carcinoma, Hodgins, leukemia, etc., (myelophthisic anemia) can be temporarily relieved with irradiation. The depression of all cellular elements, such as is found in aplastic anemia, is usually a hopeless situation, which can only derive temporary benefit from blood transfusions.

The hemolytic anemias, as shown by the preceding classification, can be grouped into the intrinsic and extrinsic varieties. Congenital hemolytic jaundice is an hereditary disease of blood characterized by red blood cells which have greater thickness than normal cells. These cells are called spherocytes, and according to Haden,[15] are less resistant to hypotonic salt solutions as less swelling can occur before they burst. The illness is characterized by hemolytic crises which result in a tremendous drop in the red blood cell count. Transfusions must be resorted to though reactions are frequent as the donor cells may be hemolyzed. Splenectomy is the procedure of choice and is followed by a relief of the anemia, though spherocytosis continues.

Sickle cell anemia is a blood disorder common to the negro race, characterized by sickle cells, or cells which assume a crescentic shape when exposed to an atmosphere of low oxygen tension. The illness is progressive and fatal, and only temporarily benefitted by blood transfusions. Haden[15] believes that early cases can be improved by splenectomy.

Mediterranean anemia is an hereditary disease of the blood found in people who inhabit the shores of the Mediterranean Sea, and in the descendents of these people. The disease varies from an individual who is quite normal except for minor blood abnormalities, including an increased resistance of the red blood cells to hypotonic saline solution, to one who has mongoloid facies, a severe anemia, splenomegaly, and bone changes. Target cells may be numerous in this blood disorder. There is no specific treatment. Many cases are mild and need no treatment, while others, the type commonly seen in children, will require the usual palliative measures of blood transfusions, rest, and adequate diet.

Paroxysmal nocturnal hemoglobinuria is a rare type of hemolytic disease in which the hemolysis occurs chiefly at night and seems to be related to a dro pin the pH of the blood. The family history is negative. The night urine is dark, while that voided in the day is normal. The fragility test is within normal limits. The outlook is hopeless. Transfusions may be used with temporary benefit, though they are usually attended by severe reactions.

Paroxysmal (cold) hemoglobinuria is a disorder occurring in syphilitics in which exposure to cold causes hemolysis and hemoglobinuria. The anemia is seldom a problem. Treatment for syphilis is often followed by disappearance of the hemolytic reactions.

March hemoglobinuria is not accompanied by anemia. Hemoglobin is found in the urine after a long march or a long distance run. The condition subsides spontaneously with rest.

Those hemolytic anemias which have external causes require the removal of the hemolytic agent for relief. In the case of malaria, atabrine and quinine are used. In streptococcal and staphylococcal infections, penicillin and the sulfonamides are the drugs of choice. Hemolytic anemias which occur in patients during the administration of sulfonamides will usually respond favorably on withdrawal of the drug, though transfusions may be life-saving in the early stages of the illness. Lead anemia is mainly a matter of recognition. The anemia is not severe and is relieved by removal of the

cause. Poisoning with the fava bean may produce a severe hemolytic anemia in sensitive individuals which may result in death. Transfusions as an emergency measure is indicated. An appreciation of the cause and the removal of this agent from the diet is of course a necessity.

CONCLUSIONS

Chronic anemias are often a problem in diagnosis and may require all available clinical and laboratory skill to determine the cause.

In some anemias, such as pernicious anemia, sprue, and the iron deficiency anemias, the results of treatment are most gratifying. In others, such as paroxysmal nocturnal hemoglobinuria and sickle cell anemia, there is little to offer the patient except supportive measures. In the anemia of carcinoma, and Hodgkins nothing can be done, as it represents a concomitant of a progressive and fatal illness.

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DISCUSSION

Dr. Johan T. Peters (New Orleans): In some of the recent editions of textbooks the blood findings in pernicious anemia are called "macrocytic normochromic", instead of the older term "macrocytic hyperchromic" which is used by the great majority of the hematologists over the whole world. The new term, recommended by Wintrobe, because the mean corpuscular hemoglobin concentration is normal in pernicious anemia, seems to be not only confusing for the students but also wrong. The prefix "hyper means only a larger amount and does not mean a higher concentration of hemoglobin. The old term "hyperchromic" was chosen because in pernicious anemia there is a larger amount of hemoglobin in the red cells, as expressed in the increased mean corpuscular hemoglobin.

Dr. Voorhies (in closing): According to Wintrobe the macrocytic anemias are characterized by the presence of many large cells adequately filled but not overloaded with hemoglobin. Because of the presence of many large cells the amount of circulating hemoglobin is higher than we would ordinarily expect from the degree of anemia present.

As is often demonstrated by pernicious anemia in relapse the high percentage of macrocytes makes life possible in the presence of a severe anemia.

RADIATION TREATMENT IN CANCER*

STAKELY HATCHETTE, M. D.

LAKE CHARLES

The use of x-rays in the field of diagnosis is familiar to everyone, but the uses of x-rays and the gamma rays of radium in the treatment of malignant disease and allied conditions are not so well understood by the average physician and it is the purpose of this paper to try to explain the reasons for using radiation therapy and the results which are obtained.

A brief review of the physical properties of x-rays and gamma rays might be helpful in understanding what they are and how they obtain their effects. Both of these agents are very similar to ordinary visible light in most of their properties and occupy their positions as wave bands in the electro-

*Read before the Sixty-seventh Annual Meeting of the Louisiana State Medical Society, New Orleans, May 14, 1947.

magnetic spectrum just as ordinary visible light rays do. The differences between these rays and light rays with which we, as physicians, are concerned is that they are invisible, are not capable of being perceived by our other senses, are destructive, and cannot be deflected.

The electro-magnetic spectrum is composed of a number of different bands of wave lengths. The longest waves are the wave lengths used for regular radio broadcasting. The next longest group is the band of waves used in short-wave radio broadcasting. This is followed by the band of infra-red rays which has medical uses and with which you are all familiar. Shorter than these, and forming a very narrow band of waves, comes the group of waves forming the visible light spectrum. Then comes the relatively broad group of ultra-violet rays which is the first form of light (or wave groups) capable of causing injury to living tissues by virtue of their powers of penetrating the uppermost layers of tissue and causing injury to the deeper layers. Their penetrating powers are not great, being only sufficient to go one or two millimeters beneath the surface, but each of you who has had a severe sunburn is familiar with the fact that ultra-violet rays can cause damaging effects to the skin. The next shortest group of rays is the band known as Grenz rays. These are more penetrating than ultra-violet rays and have been used most in the treatment of certain skin diseases. These wave-lengths are frequently thought of as the very longest group of x-ray wave lengths and they are usually generated at around 35,000 volts from a tube with a special window to permit the wave-lengths to pass through. Then comes the band of relatively long waves which are used in fluoroscopy and in making of x-ray films. These are followed by the group of even shorter rays used in x-ray therapy and these, in turn, by the gamma rays of radium which are the shortest rays used in medicine. The very shortest group of all is the group known as cosmic rays. Very little is known of them and of their effects so that they are of no known importance to medicine at this time.

Beginning with the ultra-violet wave band, as the various types of rays become shorter, they become more penetrating. As they become more penetrating, they become more destructive to living tissues. This being true, it may be said that x-rays and gamma rays are forms of light and that they are similar to light in most of their properties, but that they differ from ordinary visible light in that they cannot be perceived by any of the senses and they are destructive to living tissues. These destructive effects cannot be too strongly emphasized and this must be kept constantly in mind in working with these rays, no matter whether they be used in diagnostic procedures or in the administration of treatment. No matter how small the amount of either x-rays or gamma rays be given, and no matter whether they be used for fluoroscopy, the making of films, or the administration of treatment, it must be kept in mind that a certain amount of injury is given to the living tissues, even though it might be infinitesimal, every time they are exposed to the rays. Soon after the discovery of x-rays and radium it was found that both of these agents had a way of causing cumulative effects in the tissues and that repeated short exposures to the rays could cause destruction of living tissues just as effectively as too long a single exposure. This came about, as might be suspected, from the overenthusiastic use of these rays by the pioneer workers in making their fluoroscopic observations, x-ray films, and their administrations of the rays for the purposes of treatment.

From these observations the principle of administering multiple fractional doses of the rays for the purpose of treatment arose.

In studying the effects of radiation upon living tissues it has been found that normal, mature living cells have a greater resistance to the destructive effects of x-rays and gamma rays than do cells which are in the process of division and reproduction, and that the more embryonic the cell types ex-

posed to the effects of the rays the greater are the destructive effects of the irradiation. For this reason, it is helpful to know the degree of embryonicity of the cells in a growth to be treated, and it is because of the fact that these rays are more lethal to cells of an embryonic type than they are to mature cells that the principle of administering them to cancerous growths arose. This fact makes it possible to give a lethal dose of rays to a cancerous growth which is surrounded by normal tissues, yet give a dosage which is short of the amount required to cause destruction of the normal tissues surrounding the growth, and, while it is frequently necessary to give enough radiation to damage the normal surrounding tissues rather severely, it is the object of the radiologist to destroy the abnormal growth but give an amount of radiation insufficient to damage the normal tissues beyond their abilities to recover from the effects of the rays and to heal over the area in which the abnormal growth was present. The normal tissues are practically always irradiated along with the cancerous lesion present within them, or around it, for cancer has a way of invading the lymphatics and blood vessels in its outward extension and these invasions of tissues which seem normal to the unaided eye must be destroyed if it is to be hoped that the patient can be freed of the disease. Not only is it necessary to irradiate the tissues in the immediate vicinity of the new growth, but it is frequently necessary to irradiate tissues some distance away in order to destroy possible extension along the lymphatic drainage tracts and in the regional lymph nodes. This is the reason for giving irradiation over the entire lower abdomen in the treatment of carcinoma of the cervix or body of the uterus; the entire anterior chest wall of the hemithorax and the axillary and supraclavicular areas of the involved side in cancer of the breast, or the submandibular and cervical areas in treatment of cancer of the lower lip.

Because different normal tissues have different degrees of sensitivity to the effects of x-rays and gamma rays, and because it is sometimes found that it takes a greater amount of irradiation to destroy a new growth than it does some neighboring normal tissue of another type which has to be included in the field of irradiation, it follows that new growths in some areas are best treated by some other type of therapy. For instance, the reproductive portions of the ovaries and testes are very sensitive to the effects of irradiation and are quite easily destroyed, so that irradiation in individuals still in their active sexual lives should be given with extreme care to avoid over-irradiation of these organs, or some other type of treatment may be advised. Lymphatic tissues are next most easily destroyed so that it may be advisable under certain conditions to avoid irradiating the spleen. What is true of these two types of tissue holds for all of the other tissues of the body, but not to such great degree, so that there is not so much objection to directing a beam of x-rays or gamma rays through other types of tissues. These different degrees of sensitivity of the tissues should be kept in mind, however, in planning a course of x-ray or radium therapy to be given to a patient.

One other thing to be considered is that for reasons not yet completely understood, cancer of the gastrointestinal tract—other than the mouth and anus—seems to have quite a high resistance to the lethal effects of the rays and it takes about as much radiation to destroy them as it does to destroy the normal intestinal mucosa. Because of this, cancerous growths of the gastrointestinal tract are not treated by irradiation, but must be treated by surgery instead.

Most malignant growths, however, are capable of being treated by x-ray, by radium, or by a combination of the two. Operation is frequently necessary and, in the opinion of many radiologists, is best done *after* a certain amount of irradiation is given. The reason for this feeling is simple and logical. Because malignant growths show a tendency to early extensions into the lymphatics and blood vessels of the involved area the radiologist feels that simple surgical excision of cancerous growths is

not enough and that, in many cases, both irradiation and operation are best used together if the patient is to be given the greatest chance for recovery. For instance, in the treatment of carcinoma of the breast many radiologists believe that if approximately 1000 r of deep x-ray therapy be given to the involved breast and to the regional lymph nodes and the lymphatic and vascular drainage tracts *before* operation, enough irradiation will have been given to inhibit the rapidly growing cells about the edge of the cancer and to render them less likely to survive in the event some became dislodged at operation and be taken into the lymphatic or vascular circulations to metastasize elsewhere, or in case a few cells be left in the operative field to recur at some future time. The dosage suggested above has seemed sufficient to reduce viability of cancer cells yet it is not enough to cause the blood vessels to become congested and thereby cause the surgeon to have a more difficult, more bloody operation than he would have if nothing had been done in the nature of preoperative therapy. This should be followed by a sufficient amount of postoperative irradiation to complete the destruction of any cancer cells that might remain in the operative field and which may have invaded lymphatic and vascular tracts and lymphatic nodes draining the area. Preoperative irradiation in some degree would seem only a reasonable precaution for no matter how gently an operation be performed, there is some degree of trauma in the handling of tissues which is connected with it and which is likely to dislodge cancer cells. Then, too, the sharpest edge of the scalpel is no smoother than the edge of a saw when viewed under the microscope, and the numerous tiny cracks in the edge of the blade are more than capable of picking up small groups of cancer cells which may be transported elsewhere or which may be washed out of a crack into a blood or lymphatic vessel when one of these is cut into by the knife edge. Preoperative irradiation, if given in the suggested amount, will materially lessen the chance of any of these small groups of cells being able to survive if they be taken to other locations.

Other radiologists believe that a full dosage of irradiation should be given to a patient with cancer of the breast and that it is a wise precaution to have the breast removed after the radiation reaction has completely subsided. This is based on the fact that there are few, if any, radiologists who have not given a cancerous breast all of the irradiation the skin would stand, had a surgeon remove the breast after the reaction had completely subsided, and then had the pathologist to report that, while the original biopsy showed a cancerous lesion to be present, the breast contained nothing suggestive of cancerous tissue following its removal. There are enough breasts, however, which will show a few cancer cells trapped in scar tissue to make most radiologists feel that removal of the breast is necessary in all cases except those in which operation would endanger the patient's life. In cases where only radiation can be given, many will remain clinically free of disease until death comes from some other cause.

In treatment of carcinoma of the cervix of the uterus a course of deep x-ray therapy should be given first and should be administered through multiple ports over the lower abdomen. The reason for beginning the treatment with the deep x-ray therapy is the same as the reason for giving deep x-ray therapy preoperatively to a woman with carcinoma of the breast; that is, to destroy the rapidly growing edges of the malignancy and to kill any groups of cells which may have extended into the lymphatics draining the area. It will be found that the use of the x-ray treatment first will reduce the size of the growth materially and will greatly lessen the possibility of metastases, and will make it far safer to dilate the cervix sufficiently to permit the insertion of the radium applicator. In this connection, the practice of using a Goodell or Wylie type of cervical dilator cannot be too strongly condemned in dealing with a carcinomatous cervix. While they may be entirely satisfactory in dilating a cervix with non-malignant disease, they cause

tearing of the blood vessels and lymphatic channels and the breaking off of numerous small emboli of live cancer cells which may be washed into these two general circulations and which may easily result in metastases elsewhere. The use of a graduated set of dilators such as the Hank type is a far safer procedure, for dilation with this type of instrument is accompanied by far less maceration of tissues, far less tearing of the blood and lymphatic vessels, and is far less likely to result in metastases. After the course of deep x-ray therapy is given, it will sometimes be found that there is no visible or palpable evidence of the cancer remaining in the cervix. In any case, the size of the growth will be found to be arrested or reduced. This is the time for introduction of the radium to obtain the intense local effects of the rays and to destroy any cells of the original lesion which may still remain in the area. The use of these two agents is usually all that is necessary to eradicate the disease if it can be diagnosed in the earlier stages when it may still be classed as curable. If not diagnosed until it has spread beyond the cervix, the patient may still be able to obtain considerable relief and prolongation of life if given deep x-ray therapy and, in a few cases, clinical cures of the disease may be obtained for periods of five years or more. An operation as a means of treatment for carcinoma in this location has been generally discouraging and has been more or less discontinued except for a few localities.

As mentioned above, x-ray should be used first in the treatment of most malignant disease in which irradiation is to be used. The reason for this is simple. The x-ray tube is usually placed at a considerably greater distance from the lesion to be treated than would be the case if radium were used. As a result of this, the softer radiation which emerges from the tube or which may be set up in the filters used is absorbed by the air through which it passes and does not reach the patient in any great amount. This being so, only the more penetrating, hard rays are administered and these pass into the skin and through the skin to the depth desired to bring about the changes in the lesion being treated. Thus, in the treatment of carcinoma of the uterine cervix, x-radiation is administered through ports over the abdomen and back and passes through the skin to liberate its effects deep in the abdomen at the location of the cervix. Radium could be used similarly if there were enough radium in the hands of the average radiologist to make up a radium bomb of sufficient strength to approximate the amount of time required to administer x-rays, for radium liberates some rays so hard and penetrating that it requires approximately nine inches of lead to stop them. There are relatively few such rays in a beam of gamma rays, however, as compared to the number of longer, less penetrating rays which are present, and very few radiologists have more than a few hundred milligrams of radium to work with so that the average radiologist has to use the radium available to him in the way it will be most effective; that is, he uses the relatively small amount he has at distances varying from contact up to only a few centimeters and must necessarily use the longer rays low penetrability as well as the very short rays of high penetrability in giving his treatment. This being so, the effects obtained are largely localized to the immediate area treated, are greatest on the surface treated, and show a rapid decline in their effects as the distance from the surface increases. For instance; if radium be administered to the skin surface from a distance of 1 centimeter and the effect upon the skin be classed as 100 per cent effective, the decline in its intensity is so rapid that it would be only 25 per cent effective at a distance one centimeter beneath the skin, in the cervix, and at other locations where intense local effects are desired and where it is capable of being applied; but if a lesion has reached any appreciable size it must be treated by x-rays first so that its size may be reduced to the point where radium will be effective and, at the same time, the use of x-rays first does sufficient damage to the growth to render the possibility of metastases less likely in the event

that the tissues will have to be traumatized by dilatation or by surgical excision of a growth.

It is not the intent of this paper to list the various locations upon and within the body where radiation therapy is used effectively, but it is presented in the attempt to explain what radiation does and how it may be useful in the treatment of malignant disease. The conditions mentioned above were selected mainly because of the fact that malignant disease in these locations is so common as to be familiar to all, but what has been said of them applies more or less to the treatment of cancer in whatever location it may be found.

CONCLUSION

It is hoped that the explanations given have helped to clarify some of the confusion about the treatment of cancer by means of radiation, that they will explain why x-ray reactions must be expected if adequate treatment be administered, that they will help the physician in deciding what type of treatment should offer best results to his patient in order that there will be less delay between the diagnosis and the beginning of treatment, and that it is often necessary to use more than one form of treatment if the patient is to be given the greatest chance of a cure of the disease.

Dr. Garcia: (New Orleans): I think Dr. Hatchette is to be highly commended for his clarity and his lucidness in giving us an insight into the manner by which radiation becomes effective in the treatment of cancer. I am also impressed by the range of subjects that he covered in such a short time. For instance, he has given us some information about the physics of radiation, about the reactions of living cells to radiation, about the various explanations for such biological effects and about problems involved in the treatment of carcinoma of the cervix and of the breast.

It must be acknowledged that the full benefits of radiation therapy have not been realized, mainly due to the scepticism engendered by lack of knowledge regarding its true scope. The action of radiation is complex and any effort intended to clarify it is praiseworthy. With greater understanding will come greater clinical application. More lives will be saved by taking full advantage of all therapeutic measures useful in controlling cancer. For forty years there has been an intensive campaign of public education emphasizing the need for early diagnosis. This should be continued, and if successful, perhaps another generation of physicians will see a preponderance of favorable lesions with high rates of cure. But the bulk of the cancer material we see today is advanced, much of it completely hopeless; consequently, we should place great stress on the efficient employment of both surgery and radiation. It seems justifiable to say that considerable improvement over the results currently obtained can be achieved by increased efficiency in the use of recognized methods. I think Dr. Hatchette has been helpful in our progress towards this goal, by showing us how radiation acts.

There is, of course, a great deal of mystery as to why some tumors react well to radiation and others do not. Intensive research is being conducted of the intracellular chemistry of cells exposed to radiation and valuable information has accumulated. For instance, an enzymatic disturbance occurs, which can be detected by suitable analysis after test doses of radiation, and which may prove dependable in predicting the response of individual tumors. From the practical standpoint, however, w need not wait for such refinements, since we know well that radiation can contribute substantially to the salvage in certain forms of cancer. Furthermore, a better understanding of technic has brought standardization comparable to the standardization of surgical procedures for the major forms of cancer. As a consequence, favorable results can be obtained with consistency.

The question of combining surgery and radiation is constantly debated. Dr. Hatchette mentioned the administration of x-ray in cancer of the breast, prior to and after surgery. Arguments on this point have appeared in the literature for the past twenty-five years and entirely contradictory opinions have been expressed, principally because theories rather than facts have been used as the basis for a decision. We feel that there is conclusive objective proof that the combined treatment yields superior results in cancer of the breast. This can be demonstrated by comparison of five year survival rates in large groups of cases. Dr. Hatchette included carcinoma of the cervix among the conditions that he mentioned. I believe that in all stages of this disease the treatment of choice is radiation therapy, and we might add that the same holds true in other locations, as in intraoral carcinoma and carcinoma of the skin. Radiation efficiently administered will give up the best results in the frequent tumors of these sites. I wish to congratulate Dr. Hatchette again on his paper.

Dr. Peters (New Orleans): What has been your experience with it, whether due to radiation before the operation or not?

Dr. Hatchette (in closing): I do not think that radiation is of help at all. It has been disappointing in my hands to attempt to do much for cancer of the gastro-intestinal tract. I have had no luck

in treatment of cancer of the lung in preoperative radiation. I feel that the patient's chance of survival depends entirely upon a very early diagnosis in these two locations, and getting that patient to the surgeon as fast as is possible for a complete removal of those involved areas; but in cancer in almost any other location, I would say, "Yes," that preoperative radiation is definitely helpful. If nothing else, it will tend, I believe, to prevent spread of the disease and usually it will make the lesion somewhat smaller, so that the surgeon is going to have an easier job. I know many of them feel that if we give preoperative irradiation, we are going to make the operation a much more difficult thing for them. I believe we can give a helpful amount of preoperative irradiation without, necessarily, giving a sufficient amount to cause congestion of the blood vessels locally, and thereby cause a much more difficult operation when the surgeon does get to it.

Does that answer your question?

Dr. Peters: Yes.

FUNCTIONAL CONDITIONS OF THE NOSE*

JOSEPH STAMM, M. D.

NEW ORLEANS

One of the admitted weaknesses of the average American is a propensity for discussing his symptoms and illnesses and, with little or no urging, to supply his own diagnosis. It is amazing how many people complain of "sinus trouble", "sinus headaches" and "post nasal drip". What is more astonishing is that many patients with such complaints are found to be free of organic disease when examined. Unfortunately, the layman is not alone guilty of presumptive diagnoses of organic nasal or paranasal sinus disease. During the War, in Europe, all hospital trains or convoys could be counted on to contain many cases of supposed organic nasal or sinus disease which could not be substantiated, yet all of these cases were evacuated to the zone of communication by physicians.

In a recent article, Dr. Arthur Proetz classically describes his reaction to the symptom of postnasal discharge:

"To the most recent of those repulsive minor medical bugbears with which the American public delights in saddling itself, it has given an equally repulsive name: "postnasal drip". The term leaves nothing to the imagination. The symptom, on the contrary, leaves everything, which constitutes its chief menace. It brings up the rear of quite a procession of fearsome public enemies: Halitosis; Barber's itch; B. O.; "strep" throat; "athlete's" foot; and some others. Where the term arose I am unable to discover. Overnight the laity taught it to the doctors in hushed and often despondent tones. One hears it a dozen times a day, stigmatized usually with one of those clichés reserved for incurable diseases."

Obviously there is not such widespread organic disease of the nose and sinuses as the frequency of symptoms might indicate. The answer apparently lies in the fact that many of these self-diagnosed diseases are, in reality, an exaggeration or disturbance of normal functional processes.

Briefly, there are three primary functions of the nose: (1) it accommodates the olfactory organ; (2) it conditions the inspired air to the requirements of the pulmonary surfaces by warming, moistening and filtering it; and (3) it cleanses itself of the foreign material which it has extracted from the air. This last named function is at least as important as the rest in health, and is much more so in the control and eradication of infectious disease. In other words, it is the function of the mucus to carry off pathogenic organisms and foreign matter. The excessive mucus or "drip" frequently complained of is evidence in itself of protective activity on the part of the body.

Some of the factors which contribute to abnormalities of nasal function are:

(1) Allergens—inhaled or ingested
(2) Physical conditions
    (a) Central heating
    (b) Thermal changes
    (c) Changes in atmospheric pressure—flying, etc.
(3) Excessive and prolonged medication
(4) Emotional and metabolic disturbances

*Presented before the Sixty-seventh Annual Meeting of the Louisiana State Medical Society, May 14, 1947.

(5) Endocrine disturbances
(6) Irritants
  (a) Tobacco smoke
  (b) Fumes
  (c) Dusts
(7) Alcohol

This is not a complete list. It can be seen at a glance that several factors, contributing to disturbances of function may be present in the same individual. For example, a patient with nasal allergy is frequently guilty of excessive medication in the form of nose drops, sprays or inhalers; and, naturally, his physical surroundings are dynamic.

The diagnosis of functional disturbances of the nose and sinuses is not attended with marked difficulty. Organic disease is first ruled out by means of a careful history and physical examination. Cytologic studies of the nasal secretions are of inestimable value, especially in allergic conditions. Such examinations may be made in ordinary office practice with the expenditure of little additional time and energy.

Because of the prevalence of allergic rhinitis and sinusitis, it is deemed wise to discuss the subject briefly, particularly since some advances have been recently made in therapeusis of these conditions. Hansel describes two major categories in relation to otolaryngology.

Hay Fever: The history, clincal findings and cytologic picture make the diagnosis fairly easy. Hyposensitization to offending allergens has been a time honored form of therapy. With proper deference to our colleagues, the allergists, the results have not been uniformly successful. The newer antihistaminic drugs have proven, in many cases to be a valuable adjunct in the control of acute symptoms.

Perennial Nasal Allergy: Synonyms for this condition are vasomotor rhinitis, hyperesthetic rhinitis, etc. Many persons with this condition manifest no seasonal hay fever symptoms, although they may occur in the same individual. These patients occasionally respond well to allergenic hyposensitization, especially to dust extract. McElin and Horton have published encouraging results with the use of the antihistaminics in this condition.

Any discussion on functional disturbances of the nose would be incomplete without some mention of the excessive use of nose drops. The regrettable practice of continuing the doctor's, or druggist's prescription—intended for a passing ailment—prolongs many nasal symptoms. Drops which act as constrictors cause overventilation and drying. Most constrictors, despite much literature to the contrary, have a secondary reaction of vasodilation which causes congestion and altered secretion. Numerous reports are appearing in the literature of dependency upon the more powerful of the constrictor type of drops. The oily nose drops containing menthol, eucalyptol and similar drugs are sources of constant irritation and inflammation when their use is prolonged.

CONCLUSIONS

1. A great number of people who believe themselves afflicted with serious organic disease of the nose or paranasal sinuses, have only mild or disagreeable functional disturbances.

2. Proper recognition of these conditions with an attempt to eradicate the underlying cause is the desirable method of approach.

3. Cytologic examination of the nasal secretions is of value in diagnosis.

4. The newer anti-histaminic drugs appear to offer some help in the treatment of nasal allergies.

5. The prolonged or excessive use of nose drops or sprays is to be discouraged.

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DISCUSSION

Dr. W. A. Wagner (New Orleans): I do not want to discuss this paper from the standpoint of physiology, primarily because I am not a physiologist and secondly I could not because it has been such a splendid and thoroughly well presented subject that it leaves little for any physiologist to

discuss. I would like to say a few words from the standpoint of the clinician. As a rhinologist I think it is awfully hard to discuss nasal function without discussing the nasal adnexa. The nose, there is no question of a doubt, is the receptive for the drainage of the sinuses. I remember Servell came out some years ago and described the exacerbation of a chronic sinusitis as a false cold and there was quite a lot of discussion because of misunderstanding but I think he was right when he said "false cold", and that occurred because the nose was the reservoir of drainage of its adnexa, the sinuses. These is no question of a doubt in my mind that the physiology of the nose is altered by the pathologic condition of the sinus. That is confusing to the general practitioner and that is the reason I rise to mention this. When the nose becomes acutely inflamed as in acute rhinitis and acute sinusitis recuperation occurs because of its physiology. The nose having large drainage space between the turbinates and anteriorly and posteriorly, is able to recuperate but the sinus fails in many instances because of such altered physiology to which is added a deviated septum or enlarged turbinate which obstructs the small ostium. This altered physiologic function exists within the nose and is aggravated during infection by pathologic condition in the sinus. It is true that often one can not demonstrate to his own satisfaction physically that such a sinus infection exists and it is there where further study is imperative and unless such a sinus is properly studied by cytologic studies no one can determine the existence of such a sinusitis.

From the standpoint of diet there is no question of a doubt that diet has much to do with the appearance and physiology of the nose. Jarvis, for example, brought to our attention, the fact that a very highly acid diet had altered the physiology of the nose due to altered pathology of the mucosa. That acid diet produced a dry, red looking nose and there was no question of a doubt about that.

Now the question of postnasal drip which we heard the author mention and quoted an authority on it. Every postnasal drip is not a dietary nose. Every postnasal drip is not a sinusitis. Every postnasal drip is not an altered physiologic nose. That nose may be pathologic and the drip may be a result thereof, but whenever a patient complains of a postnasal drip do not stop looking for the cause until you find it. I must admit I can not always find the cause but there is a cause and we should make every effort to find it. When the general practitioner gets a patient whose complaint is postnasal drip he should send that patient to a rhinologist for further investigation, particularly if unable to find some generalized altered physiologic or pathologic state. I think we can be of much service to those who send patients to us.

Dr. Stamm (in closing): I wish to thank Dr. Wagner for his discussion. We are occasionally accused, in jest, of "specializing" in either the right or left nostril. It is obviously just as impossible to disassociate the nose and sinuses. Their anatomic and functional relationships are intimate.

I have nothing else to add.

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CLINICO-PATHOLOGIC CONFERENCE*

CHARLES E. DUNLAP, M. D.†
AND
EDGAR HULL, M. D.††
NEW ORLEANS

The following is a clinico-pathologic conference which was presented before the annual State Medical Society meeting.

PROTOCOL

M. D., a colored female, aged 24, divorced, was admitted to Charity Hospital in New Orleans on August 20, 1946 and died on September 20, 1946.

History: The patient entered the hospital because of throbbing pain in the rectum of five days' duration and chills and fever for four days. She had had no rectal bleeding, abdominal pain, or previous gastrointestinal symptoms. There was no history of cough, dyspnea, sweats, palpitation or chest pain, nor of pyuria, dysuria, burning on urination, or menstrual irregularity. The patient denied venereal disease or previous illness. She had experienced occasional headaches. No family history of tuberculosis, cancer or diabetes was reported. The patient was employed as a maid in a hospital.

Physical Examination: Temperature 103.4°; pulse 90; respiration 20; blood pressure 140/70. The patient was a well developed, well nourished colored female appearing acutely ill. Eyes, ears, nose and throat were negative; head was negative. The thorax was well developed. No nodules were felt in the breast and there was no discharge from the nipples. The lungs were clear to percussion and auscultation, the rhythm and sounds of the heart normal. The abdomen was relaxed and non-tender. No masses or enlarged viscera were felt. Extreme tenderness was elicited on palpation of the perineum, just anterior to the rectum; digital examination of the rectum was very painful. On vaginal examination an exquisitely tender mass was felt in the recto-vaginal septum. This mass was opened through the rectum while the patient was in the accident room and proved

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†From the Department of Pathology, Tulane University School of Medicine.

††From the Department of Medicine, Louisiana State University School of Medicine.

to be an abscess containing some 10 c.c. of thin yellow pus. She was then sent to the ward.

Laboratory Findings: Blood: red blood cells 3.68 million and hemoglobin 9.5 gm. per cent on September 10, 1946. Red blood cells 3.9 million and hemoglobin 11.0 gm. per cent on September 12, 1946. White blood cells taken each day from September 10 to September 19 were as follows: 13,900; 6,900; 7,100; 9,9800; 6,000; 13,600; 30,300, and 48,500 on the day before death. Apparently no blood counts were made before September 10, 11 days after admission. Differential counts done by a student showed young cells believed to be of the granulocytic series making up from 14 to 95 per cent of the total white count; proportion of young cells rose terminally. The hematocrit was recorded on three occasions as 22.0, 33 and 30. Kline negative. Urine: Yellow, acid, specific gravity 1.012, albumin negative, sugar negative. Sediment, occasional epithelial cells and crystals. Only two urinalyses are recorded (August 30 and September 3, 1946). No determinations of blood urea or N.P.N. were made. Blood culture on September 13 showed *Staphylococcus albus saprophyticus*—20 colonies per c.c.

Hospital Course: The patient was given sulfadiazine gm. IV stat. and gm. 1 every four hours for 24 hours. This had no apparent effect. Penicillin was begun on September 3, 1946, 30,000 units every three hours and continued through September 15, 1946. Sulfathiazole was given from September 4 to September 10. The patient's temperature ranged in irregular fashion between 101.0° and 104.0° throughout her hospital stay. Fluid intake including numerous infusions of glucose and saline ranged from 1000 to 3800 c.c. daily, and output from 2800 c.c. to 300 c.c. Output during the last three days of life was 1000 c.c., 540 c.c. and 600 c.c. on a daily intake of approximately 3000 c.c. During the first few hospital days she vomited a great amount but had no other complaints; there was, however, no improvement in her general condition.

On September 4, crepitation was felt in the rectovaginal septum and gas bacillus infection was suspected. A transfusion of 500 c.c. of whole blood and 40,000 units of polyvalent gas bacillus antitoxin were given. The region of the abscess was again opened through the perineum, evacuating 5 c.c. of foul smelling green pus. A biopsy of the abscess wall showed acute and chronic inflammation and smears showed a "mixed infection including spore forms." Culture was reported as showing staph. aureus saprophyticus, aerobacter, fecal type strep. and aerobic Gram positive and negative rods believed to be saprophytic.

On the next day, September 5, while an infusion was being given, she had an episode characterized by dyspnea and a rapid thready pulse. On September 6 she was very short of breath, there were rales at both lung bases, and gallop rhythm was noted. On September 7, a tic-toc rhythm was present with a heart rate of 140; venous pressure on this day was 115 mm. of water. An electrocardiogram recorded on September 7 showed sinus tachycardia, small QRS complexes and low T waves in the standard leads, and isoelectric T waves in leads CF 4, 5, and 6. She was digitalized with digitoxin, and on the next day was less short of breath, the heart rate slower. Shortly after midnight on September 9 she began to complain of severe pain in the left axillary region, accentuated by breathing. A friction rub was heard in this region, and rales and dulness were detected over the left lower lobe. The pain was relieved by morphine and did not recur. An x-ray of the chest the following morning showed "shadows at both bases suggestive of scattered infiltrations." The right femoral vein was found to be indurated.

On September 11, the inferior vena cava and both ovarian veins were ligated, and the right lumbar sympathetic chain sectioned. At operation a "firm, cord-like knotty vein was felt in the region of the left uterine artery". During the week following the operation she vomited a great deal; Wangensteen suction was employed. Several transfusions were given during this period. On September 13, after receiving 500 c.c. of blood followed by 1000 c.c. of glucose in saline, marked dyspnea again appeared, which was relieved in 10 minutes after the administration of morphine and oxygen and the application of tourniquets to the extremities. A portable chest x-ray on September 17 showed "suggestive infiltration in the right upper lobe, compatible with pneumonia."

On September 19, the patient had a sudden hemorrhage of about 1000 c.c. from the perineal wound and went into shock; blood pressure was not determinable. The wound was packed and her blood pressure was restored in three hours to 108/50 by the transfusion of 1500 c.c. of whole blood. Within a few hours she lapsed into coma; temperature rose sharply to 104.0°, gasping respiration developed, and she died twelve hours after the onset of the hemorrhage.

DISCUSSION

First slide: This is the temperature curve during the first week of hospitalization. Although there are fairly wide swings, the temperature is continuous, never going below 101.0°. The swings are probably due to aspirin which she received at frequent intervals.

Second slide: This x-ray was taken on September 9, the day on which she complained of severe pain in the chest. There is no definite evidence of consolidation. Rather profuse opacity is noted.

Third slide: This is an electrocardiogram made on September 7 or 8, after the patient had complained of shortness of breath and had gallop rhythm. The T-waves are low. Leads CF4, CF5, and CF6 have practically isoelectric T-waves.

Fourt slide: This is the final chest plate and was made on September 17, three days before death. It shows a very definite opacity of upper right lung field.

Discussion by Dr. Hull: I would like first to enumerate a few unfortunate omissions in the clinical study of this patient which might lead to greater accuracy in the clinical diagnosis and which might have served as guides to more effective therapy.

The most important was the failure to examine the pus from the initial drainage of the abscess. By this omission the best chance of determining the etiologic agent was missed.

Second, no blood count was made early in the patient's illness before complications developed. Therefore, the chance of the white blood count aiding in diagnosis was greatly reduced.

Third, the unusual findings of the student's counts were not checked by an experienced hematologist; hence it is not known whether reliance can be placed upon them.

Fourth, in spite of very low urinary output, and in spite of vomiting which appeared early in the patient's hospital stay, only two urinalyses were made, both during the first few days after admission. Microscopic examination was done on only one of these. Also, in view of these findings, blood urea and blood creatinine determinations should have been made.

Fifth, the final important omission was that only one blood culture was made. Therefore, the chance of discovering the etiologic agent was decreased.

Regarding the etiology, about as far as we can go is to say that this patient's illness was of an infectious nature and it was due to an organism which is not inhibited by penicillin or sulfonamides. The patient received fairly large doses of each of these without any apparent effect. We can say, therefore, that this infection was not due to hemolytic streptococcic or staphylococcic or common pyogenic bacteria.

Regarding the anatomic diagnosis, we can say quite definitely that this patient had a perirectal abscess, and because there was no evidence of disease found in the pelvis we can say that the organism entered through the rectal mucosa or blood stream and not from the generative organs. Since the patient's fever continued uninhibited after the drainage of the abscess, it was either a septicemia from the beginning or there was a second abscess which was not drained. The second thing is that the patient had thrombosis of the right femoral and common iliac and left internal iliac veins. From the data at hand we cannot say whether thrombosis occurred primarily in the pelvic veins, due to septic thrombophlebitis, or primarily in the veins of the legs and was due to stasis rather than infection. We can be quite sure that this patient developed pulmonary complications, either embolism or pneumonia, or both. About the status of this patient's heart, I do not believe we can be certain. We can be sure that the function of her heart was embarrassed, at least on the occasion that she developed dyspnea after the infusion and transfusion, but it is impossible to say whether this cardiac embarrassment was due alone to too much salt given as saline and other sodium salts such as bicarbonate given with the sulfonamides, or whether the infection was actually complicated by acute myocarditis. The electrocardiogram is not especially helpful in saying whether or not this patient had an acute myocarditis.

From the functional standpoint, we can say that this patient suffered the deleterious effects of a severe infection, that she suffered impairment of respiratory and cardiac function, and retention of urinary constituents due to inadequate urinary output. Certainly the immediate cause of death was circulatory collapse due to blood loss.

Going into the field of speculation, we may consider the etiology in more detail. What organisms are there which are not inhibited by penicillin or sulfonamides and

which commonly cause perirectal abscess? One is the *Salmonella* group, which sometimes cause septicemia as well. In some Salmonella infections there is septicemia without localized symptoms and in some cases there is abscess formation without septicemia. I can say then that organisms of the Salmonella group could produce a clinical picture compatible with the one this patient presented. Some of the colon bacilli also produce perirectal abscess, but less commonly septicemia except as a terminal agent. Another organism is the strep fecalis. It is quite resistant to sulfonamides and penicillin. Finally, I think we should mention the tubercle bacillus. Perirectal abscess is occasionally the first manifestation of tuberculosis and it occurs not uncommonly in individuals in whom pulmonary tuberculosis has previously been diagnosed. It is not definitely known whether it is due to swallowed organisms entering through the rectal mucosa or whether the organisms reach perirectal lymph nodes by way of the blood stream, in which case it would be analogous to tuberculosis of the lymph nodes elsewhere. Could this be tuberculosis of a disseminated type with a localizing perirectal lesion? It should be mentioned as a fairly definite possibility.

Another point: was the thrombophlebitis due to bacterial invasion of veins or merely a phlebothrombosis? We cannot say, but finding the indurated femoral veins would suggest the latter. The third speculation is whether there is a relationship between the ligation of the vena cava and the massive terminal hemorrhage. Certainly ligation causes increase of pressure in the veins which drain into the vena cava. It might be considered possible that increased pressure in the veins of the perineum might have been a contributing factor in the massive hemorrhage from the perineal wound.

Next, the peculiar blood picture: Was it due to a very severe infection, or was it a leukemoid reaction to sulfonamides, or were the counts erroneous? I don't know. Another thing on which we might speculate: was this a gas bacillus infection? I think not. *B. welchii* were never isolated from the pus, and crepitation was not discovered prior to the initial drainage. Probably gas forming bacilli entered after incision,—members of the colon group, strep. fecalis, and so on.

I think I should be called upon to make a final diagnosis and I will say that this patient had an infection due to an unknown organism, most likely one of the Salmonella group or the tubercle bacillus. From an anatomic standpoint, she had an abscess in the recto-vaginal septum, phlebothrombosis, pulmonary infarction and pneumonia. Whether or not she had acute toxic myocarditis, I do not know. As regards the cause of death, I should say that the immediate cause was hemorrhage and that contributing causes were a septic state and impairment of respiratory and cardiac function.

My closing remarks will be brief. Regarding the rapidity with which the tuberculous process in this patient's lungs spread, there was an x-ray made two or three days after admission. I did not have it reproduced because it was overexposed, but one could be reasonably sure that at that time there was no extensive process in this patient's lung. The second one taken prior to patient's death showed definite opacity of the right upper lung field.

The second point that I want to reiterate is that an error of omission that prevented us as clinicians from making an etiologic diagnosis was also responsible for the inability of the pathologist at postmortem examination to determine the organism responsible for the abscess. The greatest omission was neglect to culture the pus obtained from the abscess on admission. The nature of the organism might have dictated the type of therapy.

The final point: in the therapy of mixed infections which enter the tissues from the gastrointestinal tract, it has been shown that combined therapy with penicillin and streptomycin gives results that are superior to those obtained with the use of the streptomycin or penicillin alone. If this patient had been given streptomycin as well as penicillin, the perineal infection might have

been brought under control, and almost certainly the flare-up of the tuberculosis would not have occurred.

Dr. Dunlap: When the body of this young colored woman came to autopsy it was not emaciated or dehydrated. One of the difficult tasks in the management of a long febrile illness is the maintenance of adequate hydration and general nutrition and the physicians and nurses who were responsible for this patient deserve credit for excellent general care.

Externally the body showed few signs of serious disease. The abdominal incision that was made for the ligation of the ascending vena cava was well healed and showed no evidence of infection. There was a ragged defect in the perineal skin at the site of drainage of the pelvic abscess. The defect opened into a region of necrotic friable tissue which extended into the perineal fat and communicated through a small fistula with the rectum. Further investigation of this region showed extensive necrosis of the perineal soft tissues and several small abscesses which had not been drained. On opening the peritoneal cavity no free fluid was encountered and there was no evidence of active or healed peritonitis. Healed surgical incisions were present in the peritoneum on both sides of the pelvic brim where the ovarian veins had been ligated and a larger incision in the peritoneum of the right gutter where the cecum and colon had been freed and reflected medially in the ligation of the vena cava. The abdominal viscera were not remarkable.

The left pleural cavity contained 300 c.c. and the right 200 c.c. of clear yellow fluid. Pleural effusion totaling 500 c.c. should not cause serious respiratory embarrassment but it is worth mentioning that we frequently find pleural effusions at autopsy, large enough to serve as a contributory cause of death and all too often there is no clinical record that the effusion was detected during life. This patient had no pleural adhesions and no exudate was present on the pleural surfaces. The right lung weighed 500 grams which is considerably more than normal and the left lung 320 grams which is only a little above normal. There was boggy hyperemia and edema of the bases of both lungs and a nodular consolidation 3 cm. in diameter in the right upper lobe which on external examination was thought to represent an infarct. However when the region of consolidation was opened its central portion showed extensive caseation necrosis typical of tuberculosis with a broad surrounding zone of tuberculous pneumonia. I am surprised that Dr. Hull suspected the presence of tuberculosis on the basis of such meager clinical evidence but he was certainly correct.

The heart weighed 260 grams which is within normal limits for a woman of this weight (130 pounds). No abnormalities were found grossly or microscopically in the myocardium, valves or coronary vessels. In view of the clinical findings we made a particular search for myocarditis but none was found.

The liver was dark purple, bloody and considerably enlarged, weighing 2060 grams. The gall bladder was not remarkable. Throughout the gastrointestinal tract from the stomach to the rectum there were scattered petechiae of the mucosa but the only important changes were in the rectum and perirectal tissues. The rectum was filled with dark red, clotted blood and the posterior rectal wall was friable and necrotic and showed yellow-green and black discoloration. Three centimeters above the anal sphincter a fistula extended posteriorly through the rectal wall into the region of perineal infection and necrosis. There was no gross evidence of tuberculosis in this region.

The right kidney weighed only 80 grams while the left kidney was moderately enlarged and weighed 340 grams. The right kidney was a mere shell of fibrous tissue surrounding a distended, pus-filled pelvic cavity. The kidney parenchyma had been almost completely destroyed. These changes are practically diagnostic of an old, chronic, progressive pyelonephritis. A double ureter was present on the right kidney and this congenital anomaly helps to account for the unilateral pyelonephritis. The left kidney

showed slight edema of the cortex and minimal pyelitis. Its increased size and weight are best explained as a compensatory hypertrophy which developed over the course of years as the function of the opposite kidney was gradually destroyed.

The spleen weighed 530 grams which is several times the normal weight. It was soft and hemorrhagic and contained several yellow infarcts which I am not able to explain satisfactorily. The aorta was normal. The ascending vena cava was ligated just above its bifurcation but a residual lumen 2 mm. in diameter persisted at the site of ligature. Below this level the lumen was of normal caliber and was filled with a homogeneous dark red blood clot such as one would expect to form as a result of ligature of the vessel. Pale, grey laminated clots were present at the level of the iliac bifurcation and extended for a short distance into the iliac vessels. These thrombi must have been present before the ligation since they are of the type that can form only in a moving stream of blood. No thrombosis was present in the vena cava above the level of ligation. It was particularly interesting to find that the large veins of the thigh were free of thrombi indicating that blood in these veins had succeeded in finding anastomotic channels sufficient to maintain good return flow even after the vena cava had been occluded. Both ovarian veins were ligated and thrombosed and occasional thrombi were present in the retroperitoneal pelvic veins. The intensive chemotherapy may account for the fact that no suppurative thrombi were found.

Microscopic examination of tissue from the rectovaginal abscess showed extensive suppuration and tissue necrosis. Smears and culture of this tissue showed a variety of bacteria as might be expected from the fact that there was free communication with the lumen of the rectum and the perineal skin. Careful study revealed no evidence of tuberculosis in the rectovaginal tissues but in sections of the lung there was an actively spreading caseo-nodular tuberculosis. Segments of an old fibrous capsule from a previous lesion could still be identified but the active spread beyond this capsule was not exciting an effective fibrous reaction. It seems fairly certain that the tuberculous lesion in the right upper lobe represented a reactivation of an old arrested lesion. An additional microscopic finding of some interest was extensive extramedullary hematopoiesis in the liver. Blood cell formation in the liver is normal during fetal life but hematopoietic tissue disappears from the liver shortly after birth. During periods of rapid or continued blood cell destruction such as must have occurred in this patient the liver may resume its function of blood formation. The only other microscopic finding which deserves mention was the presence of tiny, scattered focal necroses in the brain. Lesions of this character may result either from direct toxic injury or from anoxemia. In this patient either toxemia from the pelvic or anoxemia from the terminal hemorrhage could have been responsible.

In summary, I agree with Dr. Hull that the immediate cause of death was hemorrhage and shock in a patient who was already extremely ill from an uncontrolled rectovaginal abscess. Treatment was heroic but ineffectual. There were two attempts at surgical drainage of the abscess but at the time of autopsy there were several unopened pockets of pus. A large total dose of 6,560,000 units of penicillin was given and this may account for the fact that no sepsis was found except in the region of the abscess. Failure of chemotherapy to control the local lesion is not surprising in view of the mixed bacterial flora and the large bulk of necrotic tissue. Penicillin in the circulating blood is not carried into ischemic tissues in effective concentrations and in treating infections in devitalized tistue it may be wise to raise the blood level of penicillin well above the ordinary effective range in the hope that effective concentrations of the drug will seep into the infected region. Even in mixed infections

penicillin can serve a useful purpose in destroying sensitive organisms thus improving the chances that the body can handle those that are penicillin resistant.

Ligation of the vena cava and ovarian veins was probably justified in view of the clinical findings but it must have taken considerable surgical courage to operate on a patient in such poor condition. The focus of active tuberculosis in the upper lobe of the right lung had not reached large size but does serve as a reminder that reactivation of arrested tuberculous lesions may occur in any prolonged, severe illness.

I believe that this patient still had a fair chance of survival up until the time of the severe hemorrhage shortly before her death. The pelvic infection, although not controlled, had been limited. The sustained fever suggests that microorganisms were gaining access to the blood stream but they were not able to establish suppurative foci in distant parts of the body. The heroic efforts which were made to save this woman's life might well have succeeded had it not been for the unfortunate and unpredictable fatal hemorrhage.

The Journal Committee wishes to announce the appointment of an Editor for the Journal in the person of Dr. Philip Harold Jones. It is felt that with Dr. Jones' known ability, energy and loyalty to the principles of the practice of medicine and organized medicine we can be confident that the new appointee will meet the requirements and responsibilities of this important office.

Dr. Philip Harold Jones was born on February 26, 1896, in Jackson, Louisiana, the son of Annabelle Smith Jones and the late Dr. Philip Huff Jones of Baton Rouge. His family moved to Baton Rouge in 1907, where his father practiced medicine and lived a life of public usefulness until his retirement, about ten years before his death in 1946 at the age of ninety-one.

Dr. Philip Harold Jones attended the Baton Rouge High School and received his B. A. degree from the Louisiana State University in 1916. He was graduated from the Tulane University of Medicine in 1920, and was the third generation of his family, in direct descent, to receive an M. D. from Tulane; his grandfather, Dr. John Welsch Jones, in 1852, and his father, in 1878, having preceded him.

He served as an intern at Charity Hospital for June, 1920, until September 1, 1920, and left to accept an appointment as a Rhodes Scholar. He studied at Oxford University, England, for four years, and earned the degree of Ph. D. in pathology in 1924. While abroad, he also studied in the clinics of Vienna, dermatology in the Hospital St. Louis, Paris, and cardiology in the London Hospital and the London Heart Hospital. He returned to the Charity Hospital in New Orleans and finished his internship in 1925. He was House Physician at Charity Hospital from 1926 until July, 1928. In the summer of 1928, he studied psychiatry for two months at the East Louisiana State Hospital.

During the year 1928-1929, Dr. Jones was a full-time instructor in medicine at Tulane, and from 1928 to 1937, a part-time instructor. In 1937, he was appointed assistant professor of clinical medicine, and is now an associate professor. In 1929, he commenced private practice, specializing in internal medicine.

He has held various offices in the Orleans Parish Medical Society, and was president in 1945. He has also served in many capacities in the Louisiana

State Medical Society and is now a member of the Executive Committee. He is a member of the American Medical Association, the Southern Medical Association, and the Lafourche Valley Medical Society. He is a fellow of the American College of Physicians, and was certified by the American Board of Internal Medicine, July 1, 1938. He is a member of the Nu Sigma Nu and the A.O.A.

During the first World War, he was in the S.A.T.C. at Tulane University, and in World War II, he was an examining physician for the local Boards and a member of Medical Advisory Board No. 1, and assistant Chief of Medical Service in the emergency unit formed at Charity Hospital.

He is the author of numerous medical articles published in leading journals and of a series of chapters on infectious diseases published in a reference book on medicine.

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THE SITUATION IN REGARD TO NURSING CARE

The supply of professional nursing care does not meet the needs of the sick.

The situation has received attention of the American Nurses' Association and of many other organizations. The principal causes are found in the dislocations and alteration in the pattern of human effort caused by the war. Additional causes are the transition from 12 hours to an eight hour day; the increase from 1,226,000 hospital beds in 1940 to 1,738,000 in 1945, the increase in population, and the enhanced desire for nursing service under conditions of full employment.

The present number of registered nurses is 318,000 and is the largest in the nation's history. It is reported that one third of these have entered into other fields. Probably another third are employed in teaching, industrial and executive positions. The remaining third need to be supplemented by 42,000 to meet the needs of today. Where are these to come from? In 1945, for example, 1,295 schools of nursing reported a freshman enrollment of 56,567. In 1946 the freshman enrollment in 1,271 schools was only 30,899. Figures for 1947 indicate an even greater drop for that year. After a careful survey by an authoritative group it was stated that "The general belief seemed to prevail that the nursing profession is desirable but that it offers too little reward to those who practice it and too high a cost to those who need it. This is like the situation in which an irresistible force meets an immovable object. How to reconcile the two aspects of this situation is an exceedingly difficult problem."

The American Nurses' Association has a program to hold nurses in the profession and to attract prospective recruits.

"On the state level today the ANA seeks amendments to the present nurse practice acts to bring about:

"1. Uniformly high standards of preparation for professional nurses.

"2. Provisions enabling nurses licensed in one state to secure more easily registration in another, an important point in remedying the present faulty distribution of nursing services.

"3. A thorough overhauling of the system of professional nursing education to eliminate exploitation of student nurses and to give them a broader academic education as a basis for the professional nursing program.

"4. The regulation of approved schools of practical nursing, and a stricter definition of the function of the practical nurse.

"5. Compulsory licensing of both professional and practical nurses.

"On the federal level the ANA is today urging the enactment of legislative measures to:

"1. Extend to nurses the benefits of coverage under the Social Security Act, the Federal Unemployment Tax Act and the Federal Insurance Contribution Act.

"2. Extend Federal Old Age and Survivors Insurance to those nurses who are self-employed.

"3. Equalize the retirement benefits of nurse veterans of World War I and World War II.

"4. Insure to professional employees full freedom of choice in the selection of bargaining representatives."

The influence of such a program upon the total number of available nurses will be slow. To supply a part of the need the use of practical nurses is much discussed. The requirements and methods to this end are not agreed upon. In a condition where the pressure for their utilization will increase it would be well for the interested parties to formulate a program.

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THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The New Orleans Graduate Medical Assembly will have its eleventh annual meeting February 23-26, 1948. This organization which sponsors the meeting is composed of New Orleans physicians who desired to bring to the doctors of this section another annual meeting in medicine which would be informative and helpful. They are to be congratulated on the success attained. The first session was attended by 511 physicians. For the 1947 session 1022 physicians registered and over 400 were regretfully rejected because of lack of hotel space.

The coming meeting gives every promise of being attractive and successful. Papers in fourteen fields of specialization are to be presented by sixteen eminent visiting speakers. The subject matter is being arranged so as to be of value to the practitioners. It is felt that those in attendance this year, as in the past, will find that each paper gives them something to take away in the field of helpful knowledge.

This year, as last, a post-clinical tour is being arranged. Those who wish to take a vacation in the winter under conditions of agreeable company and interest can enjoy the 15 day trip by plane to the historic points in Central America. On Saturday, February 28, a party composed of doctors and their wives, will leave by Pan American Clipper for Merida, Yucatan. After a four-day visit they will fly to Guatemala City, Guatemala. While in Merida many places of medical and historical interest will be visited, including some of the best preserved ruins of the Mayan empire and civilization.

Following these delightful days of companionship and sightseeing, a medical program will be presented in Guatemala City. The sightseeing schedule will then continue in various directions from Guatemala City, the last day and a half of the trip being unscheduled to permit additional side trips or entertainment in the city as may be desired.

Departure from New Orleans will be on Saturday, February 28, and the group will return on Friday, March 12.

Details and a complete itinerary are available at the office of the Assembly, Room 105, 1430 Tulane Avenue, New Orleans.

Dr. Joseph D'Antoni, President; Dr. Max Green, Secretary; Dr. Edgar Hull, Director of Program, and other officers are to be congratulated on having planned so inviting a program.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

MEDICAL DEFENSE

From information received at the office of the State Society, it has been learned that there is some misunderstanding on the part of many members of the organization in regard to the benefits offered by the State Society through medical defense in connection with malpractice suits brought against members. In order that there may be clarification of the privileges offered in regard to medical defense there should be borne in mind two separate and distinct objects.

First, the Louisiana State Medical Society, through medical defense, undertakes to defend its members against suits, threatened or otherwise, after approval of defense by the Committee on Medical Defense. This comprises the services of a lawyer, court costs and expert witnesses if the case is brought to trial. The services of the attorney are furnished also for any deliberations or services in regard to settlement or adjustment in connection with threatened claims previous to suit. Every active member is entitled to this service with no extra cost.

Second, if a case goes to trial or if in an agreement certain indemnity is agreed upon or assessed against the doctor by the court after due trial, the State Society is not obligated to pay such indemnity. For this there has been arranged a group liability policy which furnishes the individual doctor, at a nominal cost, such indemnity in cooperation and under the supervision of the Medical Defense Committee of the State Society. To undertake to pay indemnity for judgment by the State Society would involve three problems: (a) It would be inequitable for the membership at large for the Society to assume payment of judgment amounting anywhere from $500.00 to $50,000.00 against an individual doctor because the percentage of such cases is comparatively small in relation to the total membership. (b) Obviously the finances of the Society would have to be greatly increased to take care of such a financial burden. The uncertainties of this cost would create a very unsteady situation in economic status of the organization. (c) To issue such protection in the form of indemnity to meet such judgments would make it necessary for the State Society to comply with the State Insurance laws, which would require that the organization be incorporated as an insurance company. In other words it would mean that the organization would go into the insurance business. Further it would be necessary to meet the requirements of the Social Security and Internal Revenue laws concerning such corporations.

In view of the above the State Society has endeavored to do the next best thing, by arranging with a national insurance company to handle a group malpractice policy at a reduced premium rate and the company has the responsibility of meeting the payment of judgment against any members who carry such insurance.

For information it should be stated that the State Society is very proud of the medical defense feature of the organization and particularly so because of the fact that no case ever defended by our group has ever been lost in the State of Louisiana.

Following is a letter received from the Aetna Casualty and Surety Company concerning our present group policy, which we hope will clarify some of the features of the policy which have not previously been understood by some of our members.

Dr. P. T. Talbot, Secretary
Louisiana State Medical Society
1430 Tulane Avenue

Dear Doctor:

There are several aspects of the handling of the Group Professional Liability policy for the Louisiana State Medical Society which may need some clarification. For-

merly the insurance was written by another company whose method of writing the coverage varied somewhat from that of the new carrier. However, the benefits afforded under the two contracts are basically the same.

As you know, the former carrier withdrew from the risk due to adverse experience and it was, therefore, necessary to secure another outlet with another nationally known stock company, the Aetna Casualty and Surety Company. This company has pioneered this field and is presently writing some eighty per cent of all this class of business in the United States.

In the past the rates were variable as respects the type of practice, i.e.; some doctors paid more than others due to their handling of plastic cases, etc. Under the Aetna contract all rates are uniform. If a physician handles x-ray therapy, there is an additional premium for this protection provided that such coverage is desired. It is not mandatory.

There are doubtless some companies who are now offering insurance of this class at somewhat lower rates, but that has always been the case. This is generally the case as respects Roentgenologists. However, the Aetna has the experience and facilities to properly handle this type of insurance. After all, when dealing with such intrinsic items as the reputation of a physician, it is highly desirable that the insurance company be fully competent to dispose of the matters which arise with great care.

Where two or more doctors are acting as a bonafide partnership or where they employ another doctor, then it is essential that this relationship be protected by a partnership policy. This coverage was formerly provided under one policy, including the insured doctors Malpractice insurance and the rates were commensurately higher. The fact that the Aetna provides this coverage in a separate policy may have caused some confusion; however, the provisions are practically the same. There are many legal cases which set the necessary precedence to show the need for this coverage. One doctor can be held liable for the acts of his partners simply because of the partnership relationship. Claims have arisen where one was enjoined in a suit because of the acts of his partner and yet he had absolutely no knowledge of what has taken place.

Of course, where a doctor or group of doctors may operate a clinic or hospital, it is necessary to provide for this coverage under our regular Proprietors Professional Liability policy or our Hospital policy.

It is our present intention to have a representative of the Aetna Casualty and Surety Company present at the next meeting of the Louisiana State Medical Society in Monroe, Louisiana. Any questions which you may have will be answered at that time if it is at all possible.

Very truly yours,
James H. Thomas,
Agency Supervisor

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MEDICAL CARE FOR VETERANS

Recently an appeal was made through the Journal for physicians and parish and district societies to forward to the State Society office complaints received by them in regard to the present contract for medical service for veterans with service connected disabilities, now in effect between the State Society and the Veterans Administration. There has been some attempt by the Veterans Administration to disregard some of the provisions of the contract and these are summed up as follows:

First, patients have been instructed to go to the Veterans Facilities for refill of prescriptions or for the performance of surgical procedures already instituted by a doctor in keeping with the contract: for example pneumothorax cases have been required to go to Veterans Facilities for handling.

Second, there has been an attempt made by some of the regional officers to secure participation of physicians direct with the Veterans Administration rather than as a group from the State Medical Society, which was the aim and the purpose of the contract. In conference with General Hawley, Medical Director of the Veterans Ad-

ministration, recently in Chicago, he denounced such practice and promised to have it corrected.

Third, there is some complaint that this plan, known as the "Hometown Plan for the Medical Care of Veterans" is simply a misnomer because of the fact that the only hometown medical care authorized by one of the regional offices has been examination to determine whether the patient needs hospitalization in the Veterans Facility. This, of course, is contrary to the spirit of the contract.

Fourth, there has been some slowness in payment of bills for services rendered by accredited physicians in private hospitals. This has been especially true in one tuberculosis hospital where the bills have accrued over a period of nine or ten months.

These observations are presented for information of members of the Society and in order that a proper solution may be reached for such services in the future. It was never intended by this contract for such practices to exist under the "hometown" medical service for veterans.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Jan. 12, Installation meeting, Orleans Parish Medical Society, Jung Hotel, 7:30 p. m.

Jan. 13, Special scientific meeting, Orleans Parish Medical Society, 8 p. m.
Orleans Parish Radiological Society, 7:30 p. m.

Jan. 14, Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.

Jan. 15, Veterans Administration Hospital Staff, 8 p. m.

Jan. 16, The Orleans Society of X-ray Technicians, 7:30 p. m.
Executive Committee, Hotel Dieu, 8 p. m.
Lakeshore Hospital Staff, 8 p. m.

Jan. 19, Hotel Dieu Staff, 8 p. m.

Jan. 20, I. C. R. R. Hospital Staff, 12:30 p. m.
Charity Hospital Medical Staff, 8 p. m.

Jan. 21, Charity Hospital Surgical Staff, 8 p. m.

Jan. 22, Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Jan. 26, Scientific and Executive meeting, Orleans Parish Medical Society, 8 p. m.
DePaul Sanitarium Staff, 8 p. m.

Jan. 27, Baptist Hospital Staff, 8 p. m.

Jan. 28, Catholic Physicians' Guild, 8 p. m.
French Hospital Staff, 8 p. m.

Jan. 30, New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

Feb. 2, Board of Directors, Orleans Parish Medical Society, 8 p. m.

Feb. 3, Eye, Ear, Nose and Throat Staff, 8 p. m.

Feb. 4, Mercy Hospital Staff, 8 p. m.

Feb. 5, Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Executive Committee, Baptist Hospital, 8 p. m.
N. O. Society of Anesthesiologists, Charity Hospital, 8 p. m.

Feb. 6, Foundation Hospital Staff, 8 p. m.

NEWS ITEMS

Dr. J. Kelly Stone was elected president-elect of the Society to take office as president in 1949, in balloting Saturday, December 13 at the Society's office. Dr. Stone will serve as president-elect for one year, succeeding Dr. Max M. Green, who was elected last year, and who will assume the duties of president in 1948.

Other officers elected were: Dr. C. J. Brown, first vice-president; Dr. J. O. Weilbaecher, Jr., second vice-president; Dr. John Menville, third vice-president; Dr. N. J. Tessitore, secretary; Dr. Boni J. De Laureal, treasurer; Dr. Eugene H. Countiss, librarian; Drs. E. L. Leckert and Chas. B. Odom, additional members to the board of directors.

Dr. H. Ashton Thomas, the retiring president, will also serve as a member of the board of directors during 1948.

Elected delegates to the Louisiana State Medical Society for 1948-1949 were: Drs. George C. Battalora, C. J. Brown, Donovan C. Browne, W. R. Buffington, Wm. B. Clark, Sidney M. Copeland, Lucien A. Fortier, Aynaud F. Hebert, Sam Hobson, John J. Irwin, Theo. F. Kirn, Edwin H. Lawson, E. L. Leckert, Edward Matthews, Joseph P. Palermo, Wm. H. Roeling, J. Kelly Stone, H. Ashton Thomas and J. O. Weilbaecher, Jr.

Installation ceremonies will be held Monday, January 12, 1948 following the annual banquet at the Jung Hotel.

Dr. George Burch, chairman of the Tulane department of medicine, delivered the annual lecture before the Washington, D. C. Heart Association, November 12 in Washington. Dr. Burch also served

as guest lecturer at the University of Southern California and University of Utah medical school before leaving for Washington.

Dr. W. D. Beacham, associate professor of clinical gynecology and obstetrics, was elected secretary of the National Federation of Obstetric-Gynecologic Societies. The term of office is for two years. Dr. Beacham is president-elect of the New Orleans Obstetrical and Gynecological Society.

Dr. Alton Ochsner, the William E. Henderson Professor of Surgery, Dr. Ernest Carroll Faust and Dr. Joseph S. D'Antoni of the department of parasitology took part in the Second Mexican Congress of Medicine held November 7-17 in Mexico City. Dr. Ochsner presented a paper on "Hepatic Amebiasis"; Dr. Faust spoke on "A Present Day Assessment of Tropical Diseases and Tropical Medicine in the United States and Canada" and "Possible Determinants of the Pathogenicity of Endamoeba Histolytica" and Dr. D'Antoni read a paper on "Therapy of Hepatic Amebiasis."

Dr. J. W. Atkinson recently took a post graduate course in general practice at The New York Polyclinic Graduate Medical School.

While in New York Dr. Atkinson attended the New York Academy of Medicine Program.

Dr. William B. Clark served as an associate on the American Board of Ophthalmology in Chicago, October 9-11.

Dr. Clark presented a moving picture on Onchocercasis in Guatemala on the scientific program, eye section, of the American Academy of Otolaryngology and Ophthalmology in Chicago, October 12-17.

Dr. F. L. Jaubert and Dr. Waldo Treuting participated in the program of the Louisiana Society for crippled children at their annual state convention, in New Orleans, November 22-23.

Dr. Robert A. Katz has returned from England and the Continent, where he has been for the past two months. While there, he reported on his researches on Arteriosclerosis.

On November 2 and 3, Dr. Katz presented, in Chicago, to the American Society for the Study of Arteriosclerosis, two addresses: "The Status of Arteriosclerosis in Europe"; "Intravenous Ether, A New Approach to the Therapy of Arteriosclerosis".

Dr. T. A. Watters read a paper on "Emotional Implications of the Eye" at the North Carolina Neuropsychiatric Association, Durham, October 31.

Dr. B. Bernard Weinstein presented a paper for the Alabama History of Medicine Society in Birmingham on November 7, entitled "Three Men from Padua".

Drs. Eugene H. Countiss, Isadore Dyer, C. Gordon Johnson, E. L. King, John Weed, and B. Bernard Weinstein attended the Central Association of Obstetricians and Gynecologists held in Louisville in October.

Drs. Weinstein and Weed presented a paper on "Amoebic Vaginitis" at the meeting.

At the November meeting of the New Orleans Gynecological and Obstetrical Society, at the Metairie Country Club, the following program was presented:

"Introductory Remarks" by Dr. Earl Conway Smith, president.

"Hyperemesis Gravidarium" by Dr. Curtis Lund.

"Heartburn in Pregnancy" by James H. Ferguson.

The next meeting of the Society will be held Thursday, December 11, 6:30 p. m., Metairie Country Club.

At a meeting of the National Conference of Catholic Charities in New Orleans, October 13, Dr. Edmund Connely presented a paper on "Mental Hygiene and its Place in the Community."

Dr. Russell L. Holman, professor of pathology, attended the second meeting of the American Society for the Study of Arteriosclerosis in Chicago, November 2-3, and participated in a panel discussion on diseases of blood vessels. Dr. Holman is engaged in research on the relation of diet to arteriosclerosis, financed by a grant from the John and Mary Markle Foundation.

Dr. Neal Owens, professor of clinical surgery in charge of plastic surgery at Tulane, was elected president of the American Society of Plastic and Reconstructive Surgery at a recent meeting of the group in San Francisco.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

EAST AND WEST FELICIANA BI-PARISH SOCIETY

The East and West Feliciana Bi-Parish Medical Society met in Clinton at the Baptist Church, December 3, 1947, at which time an excellent dinner, prepared and sponsored by the Clinton Infirmary and supervised by Mrs. Willie B. Hubbs, Mrs. Bentley and Miss Sue Palmer, was served. The following officers were elected by the society to serve for 1948: Dr. C. J. Wise, Angola, President; Dr. W. J. Roberts, Woodland, Vice-President; Dr. E. M. Toler, Clinton, Secretary-Treasurer. Dr. E. M. Toler was elected delegate to the State Society meeting and Dr. Glenn J. Smith was elected alternate.

Dr. Courtland Smith, of Baton Rouge, presented a paper on conjunctivitis which was very favorably discussed by the physicians present and a vote of thanks was extended him for this contribution.

At this meeting Dr. J. F. Dinan, of the East Louisiana State Hospital, was elected a member of the society and a vote of thanks was extended to the Clinton Infirmary and the Staff for the excellent entertainment and dinner.

The next meeting will be held at the East Louisiana State Hospital on the first Wednesday in March, 1948.

WEBSTER PARISH MEDICAL SOCIETY

At a meeting of the Webster Parish Medical Society on December 9 at the Minden Sanitarium, Minden, the following officers were elected for 1948. Dr. T. A. Richardson, President, Dr. E. S. Rogers, Vice-President, Dr. S. M. Richardson, Jr., Secretary-Treasurer, Dr. C. M. Baker, Delegate to State Society, Dr. W. C. Gray, Alternate. After the election Dr. W. C. Gray presented two colored movies. The first was a partial resection of the stomach and the second was a Wertheim operation. Dues for the coming year were received.

FIFTH DISTRICT ELECTS DR. COON

A meeting of the Fifth District Medical Society was held on Thursday, December 11, 1947, in the Virginia Hotel at Monroe, Louisiana. Members of the Fifth District elected Dr. H. S. Coon of Monroe as their president and Dr. Fred Marx of Monroe was named secretary-treasurer. Dr. Coon succeeds Dr. Ralph King of Winnsboro, the immediate past-president of the District. Dr. King was elected as a delegate to the House of Delegates of the Louisiana State Medical Society, scheduled to meet in Monroe in its annual meeting in April, 1948. Dr. C. P. Gray, Jr. of Monroe, President of the Ouachita Parish Medical Society. was named as an alternate delegate.

Presenting papers at the meeting were Dr. Edgar Hull of New Orleans, professor of medicine at L. S. U.—"Recent Advances in the Treatment of Heart Diseases"; Dr. Barrett Kennedy of New Orleans, associate professor of dermatology at L. S. U.—"Skin Rashes Due to Drug Administration"; and Dr. Donovan C. Browne, professor of clinical medicine at Tulane Medical School—"Value of Proctoscopic Examination".

Other speakers at the meeting included Dr. Gilbert C. Anderson of New Orleans, President of the Louisiana State Medical Society, who spoke on "Malpractice Insurance", and on "The Growth of Louisiana Physicians Service", the surgical and obstetrical care plan sponsored by the State Society. The program also included Dr. P. T. Talbot of New Orleans, Secretary-Treasurer of the State Society, and Frank Lais, Jr., Executive Director of the Council on Medical Service and Public Relations and Louisiana Physicians Service. Mr. Lais spoke on "The Proposed Public Relations Program of the Council on Medical Service and Public Relations".

SCIENTIFIC EXHIBIT

At the 1948 meeting of the Southern Medical Association, recently held in Baltimore, an exhibit entitled "Effects of Beta-radiation in Ophthalmology: Ciliary Body of Rabbits" was presented by Miss Aleta Barber, Dr. Louis Breifeilh and Dr. George M. Haik. The exhibit concerned work car-

ried out at the Louisiana State University School of Medicine.

NU SIGMA NU—RUDOLPH MATAS LECTURESHIP

The Beta Iota chapter (Tulane) of NU SIGMA NU Medical Fraternity is inaugurating an annual lectureship in honor of its most famous member, Dr. Rudolph Matas, Professor of Surgery Emeritus at Tulane and probably the most widely acclaimed living physician. This lectureship is to be known as the "NU SIGMA NU—RUDOLPH MATAS LECTURESHIP", and each year it is planned to bring some outstanding physician to New Orleans to deliver an address. The first lecture will be held on January 28, 1948 at 8:00 p. m. in the Hutchinson Memorial Auditorium of the Tulane Medical School. The organization is fortunate in being able to have Dr. Alfred Blalock, an eminent vascular surgeon and Professor of Surgery at Johns Hopkins Medical School, as the inaugural speaker. His subject will be "Surgical Treatment of Congential Cardiovascular Defects" with accompanying slides and movie film.

All members of the medical profession are cordially invited to this lecture.

AMERICAN COLLEGE OF SURGEONS

Dr. Arthur W. Allen, President of the American College of Surgeons, announces six Sectional Meetings scheduled for 1948, for Fellows of the College, the medical profession at large, and hospital personnel. Each meeting will be two days in length and will include conferences for hospital personnel as well as sessions for the medical profession. The showing of medical motion pictures will begin each day's progam at 8:30 a. m. There will be luncheon meetings each day and a dinner meeting on the first evening. The latter will be followed by a symposium on cancer. Panel discussions on scientific subjects, led by internationally known authorities in each field of surgery, will be held each morning and afternoon. The list of meetings follows:

Toledo, January 20 and 21, Commodore Perry Hotel.

Atlanta, January 26 and 27, Ansley Hotel.

Oklahoma City, January 30 and 31, Oklahoma Biltmore Hotel.

Denver, March 1 and 2, Cosmopolitan Hotel.

Minneapolis, March 15 and 16, Hotel Nicollet.

Halifax, May 17 and 18, The Nova Scotian.

Among the subjects to be discussed at the scientific sessions will be fractures of the upper and lower extremities; pediatric surgery; importance of the use of blood and fluids and of adequate nutrition in surgery; early diagnosis and proper treatment of cancer; organization and functioning of cancer clinics and cancer detection centers; intestinal obstruction; management of wounds, surgical incisions and fresh traumatic wounds; urologic surgery; plastic surgery; vascular surgery; and panel operations on elderly patients with special reference to the reduction of surgical risk.

Among the subjects which will be discussed at the hospital conference will be the increasing use of hospitals; expansion of hospital facilities; higher standards of training for hospital administrators; improvement in personnel policies; increasing cost of hospital service; better rural hospital service; coordination of hospital with other health and welfare activities in the community; Blue Cross and medical service plans; decreasing average days' stay in hospitals; participation of hospitals in cancer control; advances in physical medicine; increasing importance of chemotherapy; nutrition in relation to disease; changes in nursing service; improved status for the general practitioner; decreasing rates of deaths, infections, and complications; care of chronic and psychiatric patients; advances in professional services; medical staff organization; the professional audit; and the point rating system.

The American College of Surgeons has a fellowship of 15,500 surgeons in the United States, Canada, and other countries. Dr. Irvin Abell of Louisville is Chairman of the Board of Regents. Dr. Malcolm T. MacEachern and Dr. Bowman C. Crowell of Chicago are the associate directors. The College was founded thirty-five years ago. Headquarters are in Chicago.

SOUTHERN SOCIETY FOR CLINICAL RESEARCH

A meeting of the Southern Society for Clinical Research will be held at the Roosevelt Hotel, New Orleans January 27, 1948.

AMERICAN HOSPITAL ASSOCIATION

How gifts to hospitals can cost donors less than the apparent amount because of decreased income taxes is explained in a brochure issued by the American Hospital Association.

Charitable donations of up to 15 per cent of adjusted gross income of individuals, and up to five per cent of net taxable corporation incomes are exempt from income tax according to current laws, according to the booklet, "Your Gifts to Hospitals through Taxes."

Hospitals over the country are facing spiraling costs like everybody else," said George Bugbee, executive director of the Association, "but the care of patients can't be pared down as some commercial and personal budgets can.

"You can't go halfway on health. That is why funds for expansion and endowments, especially through the easier tax deduction method, are so important to voluntary, non-profit hospitals today.

From $185.25 saved on a $5,000 gift to $95,000 on a $250,000 donation, substantial economies can

be made, the Association states. Donations of securities and other properties can result in even greater benefits.

Savings of 38 per cent in gifts from corporations and of up to 86.45 per cent in individual gifts are possible under the present income tax regulations, the booklet points out.

STANDARDS RECOMMENDED FOR ADOPTION BY THE PHARMACOPOEIAS OF THE WORLD

At its recent sessions in Geneva, from 13 to 17 October, 1947, the Committee of Experts on the Unification of Pharmacopoeias of the World Health Organization of the United Nations, placed 244 items on its Primary List of medicinal substances. These they believe of sufficient importance for immediate attention and inclusion in a book of "Standards Recommended for Adoption by the Pharmacopoeias of the World". This Primary List will be further submitted for review to authoritative medical groups in a number of countries affiliated with the World Health Organization.

A total of 534 drugs were studied. In addition to the items proposed for inclusion in the Primary List, 89 drugs were placed in a Secondary List. The drugs in this second list were known to have some medicinal value but were believed of lesser importance and their standardization can be deferred until a later date. Among the drugs studied, 201 were dropped from further consideration on the ground that they have become obsolete and find little use in modern medical practice.

Starting with the foundations laid by their predecessor, the Technical Commission of Pharmacopoeial Experts of the Health Organization of the League of Nations, the new Committee, during the course of the recent sessions at Geneva, completed the first drafts of 72 monographs of the drugs selected for the Primary List.

During the next few months drafts of monographs will be prepared by the members of the Committee for all items in the Primary List and these suggested texts will be submitted for review to international experts in pharmacopoeial revision.

Another meeting of the Committee is proposed for early next summer, when it is hoped that the First Edition of a book of Standards will be completed.

The present membership of the Committee of Experts includes: Professor H. Baggesgaard-Rasmussen, Chairman of the Chemical Division of the Danish Pharmacopoeia Commission; Professor I. R. Fahmy, Professor of Pharmacognosy, Faculty of Medicine, Cairo; Professor E. Fullerton Cook, Chairman of the Committee of Revision of the United States Pharmacopoeia; Dr. C. H. Hampshire, Secretary of the British Pharmacopoeia Commission; and Professor R. Hazard, Professor of Pharmacology and Materia Medica, School of Medicine, Paris. Professor Hazard was prevented by illness from attending the October session. Dr. Hampshire, who had been the Chairman of the former Committee, was elected Chairman of the new Committee.

It is expected that the membership of this Committee will be increased, so that additional countries will be represented, and as wide an international basis as possible, will be provided.

LOUISIANA STATE MEDICAL SOCIETY WOMAN'S AUXILIARY

The Woman's Auxiliary desires to express their sincere appreciation to Dr. Talbot, the members of the Journal committee and to Miss Shoemaker, for their fine co-operation in again publishing in the Journal the Year Book of the Auxiliary. The Year Book is a valuable aid to the members, for which we are deeply grateful.

A big bouquet of red roses—verbal ones—goes to Mrs. Edgar Burns for her work in assembling and preparing the material for the Year Book. This was a tedious and exacting task, well done, and which it was necessary to perform during the hot summer months. We especially appreciate the effort put forth by Mrs. Burns.

The President of the Louisiana State Medical Auxiliary, Mrs. James W. Warren, attended the Fall Conference of State presidents, presidents-elect and National officers in Chicago on November 6th and 7th, 1947. Mrs. Warren commented that the conference was very helpful and inspiring.

Mrs. Warren desires to express her appreciation to the various chairmen of standing committees who have sent out such inspiring and helpful letters to parish presidents regarding the work of their departments. Each parish president is urged to see to it that these letters are read in open meetings as this is the only medium through which the chairmen can reach and appeal to the members.

Many of our auxiliaries are going forward on our new project "Preservation of Medical Cultural Items". Shreveport, Monroe and Baton Rouge have had Mr. D. W. Postell, Librarian of the Louisiana State University Medical School at New Orleans, speak before their groups on this subject. It is our plea that the Auxiliaries work actively on this project and turn in whatever material they may have to Mrs. Rhodes J. Spedale, Plaquemine, Chairman of Preservation of Medical Cultural Items.

NOTICE

The MID-YEAR EXECUTIVE BOARD MEETING will take place in New Orleans on Thursday, February 26th, 1948 and reports will be due at that time. You will be notified of the time and place about ten days before the meeting.

BOOK REVIEWS

A Primer of Cardiology: By George E. Burch, M. D., F. A. C. P. and Paul Reaser, M. D. Lea & Febiger, Philadelphia, 1947. Pp. 272. Price, $4.50.

This book contains two hundred fifty seven pages including the appendix. It is divided into five chapters.

The first chapter begins by calling the attention of the student to some of the anatomical facts concerned with the study of heart disease and the importance of the position of the heart in the thorax. There are numerous drawings throughout the book to help the reader understand the descriptions given in the text.

In Chapter II there are seventeen signs given as dependable or pathognomonic signs of heart disease. There is a short discussion of each one. In the complete diagnosis, including the functional and therapeutic classification, the authors follow the nomenclature of the American Heart Association: Heart failure—anginal—congestive. The latter divided into left, right and combined ventricular failure and their associated syndromes are described.

The discussions of the conditions are not extensive but are adequate for the purpose of this book. They are consistent with the views of most modern cardiologists.

At the end of Chapter II there are ten pages devoted to the discussion of edema, pointing out that the mechanism of cardiac edema is not fully understood.

In Chapter III heart sounds and murmurs are discussed and the mechanism of their production is described in the text and by drawings.

Chapter IV is devoted to the common types of heart disease. Heart disease is defined as any disturbance in function that disturbs the patient in any way. This includes cardiac neurosis. There are some who might object to a too literal application of the word disease. It is true that some cases of cardiac neurosis are just as disabled and many of them are harder to treat than those with organic heart disease. One of the most helpful means of handling these cases is to be able to tell them truthfully and emphatically that they have no heart disease as this term is understood by the laity. Names given to conditions by physicians have a way of finding their way into the vocabulary of the lay public and are frequently interpreted differently from that intended by the physician. So it might be best not to encourage the application of the word disease to the various types of cardiac neurosis where there is no evidence of actual disease of the heart.

At the end of Chapter IV there is a discussion of congestive heart failure which is very good and should be of interest to all.

Chapter V deals with the bed-side diagnosis of cardiac irregularities. The subject is well presented for the purpose of the book. A few minor changes in wording would be of advantage. In the treatment of premature beats it is advised to have the patient sleep longer hours, take frequent naps or rest reclining frequently. This would be well for patients with heart disease but when premature beats are the only findings the patient's activities as a rule should not be restricted. It is stated that if a late diastolic, crescendo murmur is heard auricular fibrillation cannot exist. This is true, however, if the diastolic period is short enough and early diastolic murmur may give the impression of being late and being crescendo also even when auricular fibrillation is present.

There is an appendix of sixteen pages in which the nomenclature and classification of heart disease advised by the New York and American Heart Association is reproduced and tables for heart measurements and cardiac diets are given.

One might feel that a little different arrangement of the subject matter would have been better but the book will fill its purpose well and any one interested in cardiology will find the reading of it interesting and helpful.

J. M. BAMBER, M. D.

The Foot and Ankle: Their Injuries, Diseases, Deformities and Disabilities: By Philip Lewin, M. D., F. A. C. S. 3rd ed. thoroughly revised. Philadelphia, Lea & Febiger, 1947, Pp. 847. Price. $11.00

This 3rd edition of Dr. Lewin's popular book has been enlarged by the addition of 227 pages. There have been few if any deletions, and several illustrations have been added. The additions are primarily the advancements which have been made in the methods of treatment since the last edition. They reflect the changing trends of thought brought about by the experience of orthopedic surgeons in World War II and by the introduction of antibiotics.

The same style with readable and accurate information has been retained. The addition of new material has improved the usefulness of this edition as a reference book for all physicians and surgeons concerned with treating disturbances of the feet or ankles.

R. M. KIMBALL, M. D.

Handbook of Fractures: By Duncan Eve, Jr., M. D., F. A. C. S., and Thimble Sharber, A. B., M. D. 3rd ed. St. Louis, C. V. Mosby, 1947. Illus. Pp. 263. Price, $5.00.

This handbook of fractures has been presented for the benefit of the younger members of the profession and for those who only occasionally undertake the treatment of fractures. This is an extreme-

ly practical book, with the emphasis having been placed on the details of treatment. The illustrations are well done and are adequate in number.

The author has consistently brought out the essential factors in the management of the common fracture problems with the minimum number of words. Furthermore, he has presented in detail a single method of treatment of the common fractures which is conservative, modern, and reliable. This brevity and clarity of the text and illustrations, combined with the author's "seasoned" choice of methods, makes this handbook invaluable to the student, interne, resident, and general practitioner.

R. M. KIMBALL, M. D.

Handbook of Communicable Diseases: By Franklin Henry Top, A. B., M. D., M. P. H., F. A. C. P. 2nd ed. St. Louis, C. V. Mosby, 1947. Pp. 992. Price, $8.50.

This, the second edition of F. H. Top's Handbook of Communicable Diseases is divided, as was the first edition, into three sections. The first section is concerned with general considerations relative to communicable diseases. Infection and immunity are discussed briefly, emphasizing tests for immunity and immunization procedures. Chapters on the management of communicable diseases in the home and in the hospital are included. In the second section specific diseases are discussed in a clear, concise form with the diseases classified according to the portal of entry of the causative agent. The last section presents in table form experiences of the Herman Kiefer Hospital with the common communicable diseases.

Special mention of the two chapters, written by nurses, on the management of communicable diseases in the home and in the hospital is deserved. This edition of Top's book has been much improved by the addition of chapters on fourteen diseases not included in the first edition. These include chapters on coccidioidomycosis, rheumatic fever, primary atypical pneumonia, infectious hepatitis, and infectious mononucleosis. The book is adequately illustrated with photographs in color and in black and white. Though this is not a comprehensive and exhaustive work, it is recommended as a ready reference on the more common communicable diseases.

HARRY V. HERNDON, M. D.

Cineplasty: By Henry H. Kessler, M. D., Ph. D. Springfield, Illinois, Charles C. Thomas, 1947. Pp. 201, 314 illus. Price, $6.75.

This excellent monograph by Kessler is written with well-founded authority. The subject of cineplastic amputations is a controversial one. Much interest was revived during the past war in the entire problem of amputation and prosthetic devices and again in cineplastic amputations. The scope of this monograph is not limited to cineplastic amputations alone, but considers the entire problem of the amputee, both from the physical and psychological point of view, as well as an extensive chapter on the rehabilitation of all amputees.

The sections on the history of cineplastic procedure, the physiology and anatomy, and the surgical techniques are exceptionally good. Chapter VII, which deals with cineplastic prostheses, probably does not convey the true limitations of the present prostheses which are available for such cases. The chapter on phalangization and the chapter on plastic procedures and amputations, cover the procedure of phalangization of the first metacarpal and the use of rotation osteotomy of the fingers to oppose the thumb. The Krukenberg operation, or split forearm operation designed to furnish prehensil power as well as tactile sensation, is thoroughly discussed and evaluated.

The style in which Dr. Kessler writes is extremely readable, authoritative, and quite terse. The critics of the cineplastic amputation and of Dr. Kessler's work in this field have based their criticisms on the lack of an adequate prosthetic device for the fitting of these patients. This monograph furnishes an extensive amount of material for consideration by all who are interested in amputations and the restoration and the rehabilitation of amputees.

JACK WICKSTROM, M, D.

Textbook of Medicine: By Russell L. Cecil, A. B., M. D., Sc. D., Walsh McDermott, M., and Harold G. Wolff, M. D. 7th ed. Philadelphia, W. B. Saunders Co., 1947. Pp. 1730. Price, $10.00.

When "Cecil's Medicine" was presented originally 20 years ago, it was recognized to be a most welcome addition to the group of useful texts for students. That it is now presented in its seventh edition is adequate evidence to its continued value and worth as a means of imparting clinical information.

As in the sixth edition, the text is presented in double columns. The liberal use of illustrations, and particularly the inclusion of 18 color photographs, makes the text much more readily understood. The current interest in psychosomatic medicine is reflected in a new chapter on this subject; the resurgency of organic neurology is likewise represented by new articles on hemifacial spasm, on narcolepsy and on diphtheritic polyneuritis; similarly there are several contributions on vitamin deficiencies and excesses not previously covered. Practically all of the other articles have been largely revised.

This book can be recommended to any junior or senior medical student for routine use. It will be

of service to many practitioners who wish a brief discussion of those clinical entities usually regarded as within the domain of internal medicine. While most of the discussions are necessarily dogmatic, differences of opinion are frequently admitted by the insertion of an appropriate foot-note.

The seventh edition will undoubtedly be as well received as its predecessors.

SIDNEY JACOBS, M. D.

PUBLICATIONS RECEIVED

F. A. Davis Company, Philadelphia: Textbook of Human Physiology, by William F. Hamilton, Ph. D.

Lea & Febiger, Philadelphia: Diseases of the Nose, Throat and Ear (9th edition), by William Lincoln Ballenger, M. D., F. A. C. S., Howard Charles Ballenger, M. D., F. A. C. S., assisted by John Jacob Ballenger, B. S., M. D.; The Foot and Ankle (3rd edition), by Philip Lewin, M. D., F. A. C. P., and Paul Reaser, M. D.; Surgical Disorders of the Chest (2nd edition), by J. J. Donaldson, B. S., M. D., F. A. C. S.; Unipolar Lead Electrocardiography, by Emanuel Goldberger, B. S., M. D.

Medical Center Foundation and Fund, Chicago: Ulcer, by Donald Cook, B. A., M. D.

C. V. Mosby Company, St. Louis: Handbook on Fractures, by Duncan Eve, Jr., M. D., F. A. C. S.; Synopsis of Neuropsychiatry (2nd edition), by Lowell S. Selling, M. D., Ph. D., Dr. P. H., F. A. C. P.

Henry Schuman, New York: 400 Years of a Doctor's Life, by George Rosen, M. D. and Beate Caspari-Rosen, M. D.

Charles C. Thomas, Springfield, Illinois: Practical Office Gynecology, by Karl John Karnaky, M. D.